

Cutaneous Immunology & Allergy

- Immunology of skin
 - Eczema (Dermatitis)
 - Urticaria & Angioedema
 - Drug Eruption
 - Photosensitivity
-

Eczema (Dermatitis)

References

- Serieb
- Darouzi
- Andrews
- Balogun
- E. medicine
- Derm net

Rock.

Introduction:

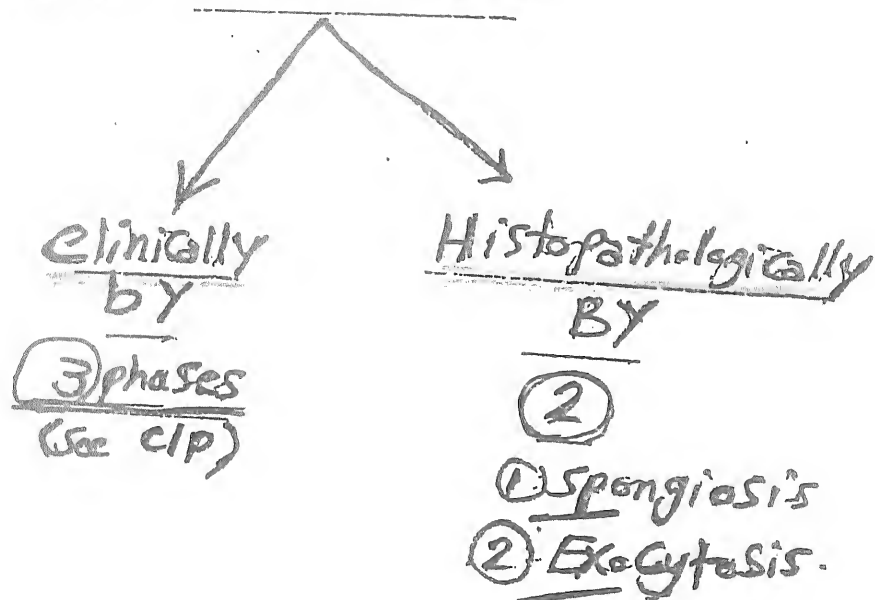
- * Def.
- * Incidence
- * Pathogenesis
- * C/P
- * Complications
- * Treatment

* definition: -

ECZema: Come from the Greek word "EKZEIN"
 فوران. It means "To Boil" or "To effervesce" ^{لغوة}

Dermatitis: ^{حرقا} Literally = inflamm. of the skin
 So it's broad (non) specific Term (all ECZ are dermatitis but ^{لغوة} not all dermatitis are ECZ.)
 But

Both words used by dermatologist to describe group of inflammatory skin disorders caused by variety of Ext. & internal stimuli That's characterized:



NB

ECZema,

بشيء دكتور أوروبا

Dermatitis:

بشيء دكتور أمريكا

Acne
 Release → perioral dermatitis

ما ينبغي
 أقول على
 Eczema.

- pathology of ECZ : (Eczema)

Perioral eczema = CD
Citrus Food / Tongue induced.

• Acute ECZ.

• Subacute ECZ.

• Chr. ECZ.

Acanthosis.
Parak.
Dermal
fibrosis.

Early: - Dermal Edema

(Dermal) - Perivascular Lymphohistiocytic infilt.

then: (Epid) ① Spongiosis

② Exocytosis

③ Blister format (Vesicle)

Spongiosis ↓↓

① Parakeratosis

② Acanthosis

Crustat (Coagulated plasma + Pyknotic Nuclei)

Epidermal → Acanthosis & Parakeratosis.

Dermis: Dermal

fibrosis (detected clinically as Lichenificat.)

NB Exocytosis is (Lymphocytes + Spongiosis) infilt. if there is:-

↑↑ Marked spongiosis + Marked Lymph. infilt.

Dermatitis [Eczema]

↓ Scanty Spongiosis + ↑ Marked infilt.

MF

psychic

ptn
يجعل على لونه
بعض المراحل الأولى
(my lichenific)

C/P of ECZ. (General Features)

Itching (Cardinal)

Ill defined (Do from papulosq. Erupt.)

has (3) phases:

• Acute

- Erythema

✓ Papulovesiculatn

- blister & ± bullae

✓ Weeping = يبكي

✓ oozing

• Subacute

- Erythema

- Papules

← Scaling & Crustat (8)

↓ Vesiculatn & oozing (ببأ حيف)

• Chr.

itching

Lichenificat.

thickening

Hyperpig.

↑↑ skin Markings

(Exaggerated)

Treatment of Eczema

Acute Weeping ECZ.

①

drying antiseptic lotions

10 mins Cool Soaks

0.65% Alum. Acetate
saline
Tap Water
K-permanganate

then

②

Smear of Cs Cream or lotⁿ

0.65% Al acetate
potters sol
مال

③

non adhesive dressings

non occlusive

Staph

موجود على

Subacute ECZema

① Cs lotⁿs or Creams

② ^{مزيل الحكة و دواء} yellow crust = Inf → Fucidate

NB. staph Colonize:-

- ① all Weeping ECZ.
- ② Most of dry ones.

to differentiate bet. Colonization & overt Inf:-

- ① Heavy Crusts
- ② Large No. Islets
- ③ CRP

staph. Inf. or colonization may aggravate ECZ. & delays Healing

See ECZema = Think Staph

فيل ريب
Staph

Chr. Lichenified ECZ

Cs Ointments

also Calcineurin inhibitors (Cs)
Ichthammol
Zinc

ولا يتركها
قوة + Cs

age site amount
50

① don't use potent Cs > Hydrocortisone

1% in

Face Flexures Infant

② don't use:

> 200 gm / W of mild potent

> 50 gm / W of mod. potent

> 30 gm / W of superpotent

أخف بقاء له على
أزود اللحية

2-3 W

then every 4 weekend

Should topical steroids be used on lesions that may be infected or colonized with bacteria?
Absolutely. Lesions get infected because the skin defense barrier is broken. The topical steroids allow it to heal, and thus the bacteria have greater difficulty infecting the skin. Such bacteria have receptors for fibrin or fibronectin that can be exposed in dermatitic, but not normal, skin.

Acute Weeping ECZ.

drying Antiseptic + Cs lotⁿ oint

Subacute ECZ.

Cs كريم

Chr. Lichenified ECZ

Cs ↑ potency
↑ moisturizing
مرهم

Classification of eczema.

According to the causative factor whether external or internal;eczema can be classified into :

Exogenous Eczemas بره	Endogenous Eczemas جوه الجسم
<p>①-<u>Contact dermatitis (CD)</u>: (حسب) Allergic CD chemical Irritant CD مع الضوء Photoallergic CD</p> <p>②-<u>Infective dermatitis</u></p> <p>③-<u>Dermatophytid</u> = (Id) reaction.</p> <p>④-<u>Eczematous PML</u> ← Sun exposure</p> <p>⑤-<u>Post-traumatic eczema</u></p>	<p>✓-Atopic dermatitis = genetic</p> <p>-Seborrhoeic dermatitis</p> <p>-Stasis dermatitis</p> <p>-Frictional lichenoid dermatitis</p> <p>-Xerotic eczema</p> <p>-Discoid eczema</p> <p>-Pityriasis alba</p> <p>-Juvenile plantar dermatosis</p> <p>-Eczematous drug eruptions</p> <p>-Metabolic eczema or eczema associated with systemic disease</p> <p>→ Hand eczema</p> <p>-Exudative discoid and lichenoid dermatitis</p> <p>-Chronic superficial scaly dermatitis</p>

NE: 1- This classification isnot absolute because of interaction between the 2 factors.

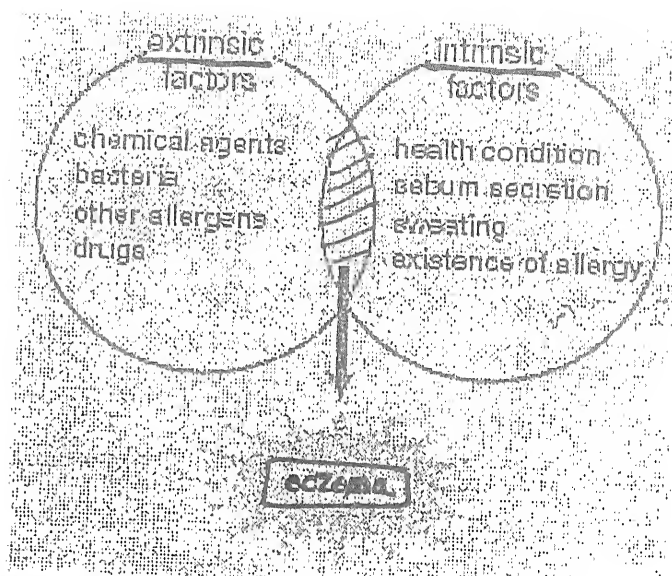


Fig. 7.5 Various factors causing eczema.
Extrinsic and intrinsic factors interact, resulting in eczema formation.

Dermatitis
Venereata

Allergic CD (ACD)

def
Incid
Pathophys.

def
Invs

def delayed Hypersensitivity reaction (Type IV Allergy)
resulting from cut. contact w specific Allergens
to w the patient develop specific Allergy.

Incid: (20%) of CD cases (ICD: (80%), ACD (20%))

Pathophysiology: Type IV Delayed Hypersensitivity reaction
w has 2 phases: - المرحلة

(الأسبوع)

1 Induction (Sensitization) phase: (10-14 ds) ^{1st exposure (2 weeks) أسبوعين}

(اليوم)

2 Elicitation (Challenge) phase: (1-3 ds) ^{الإثارة}

1 chemicals that can produce ACD: - ^{Recurrence exposure (2ds) يومين}
• ~ 3000 in No
• of small sized molecules < 500 daltons.

2 Pathogenesis: LMW Ag hapten < 500 daltons →
When contacts the skin form hapten-carrier
protein complex → Lcs display
it as complex with (HLA DR) on their
surfaces → presented to CD4 → ^{ال}
Interact w TCRs CD3 complex of CD4 → ^{ال}

3 ACD ch-by:

ميزة لها

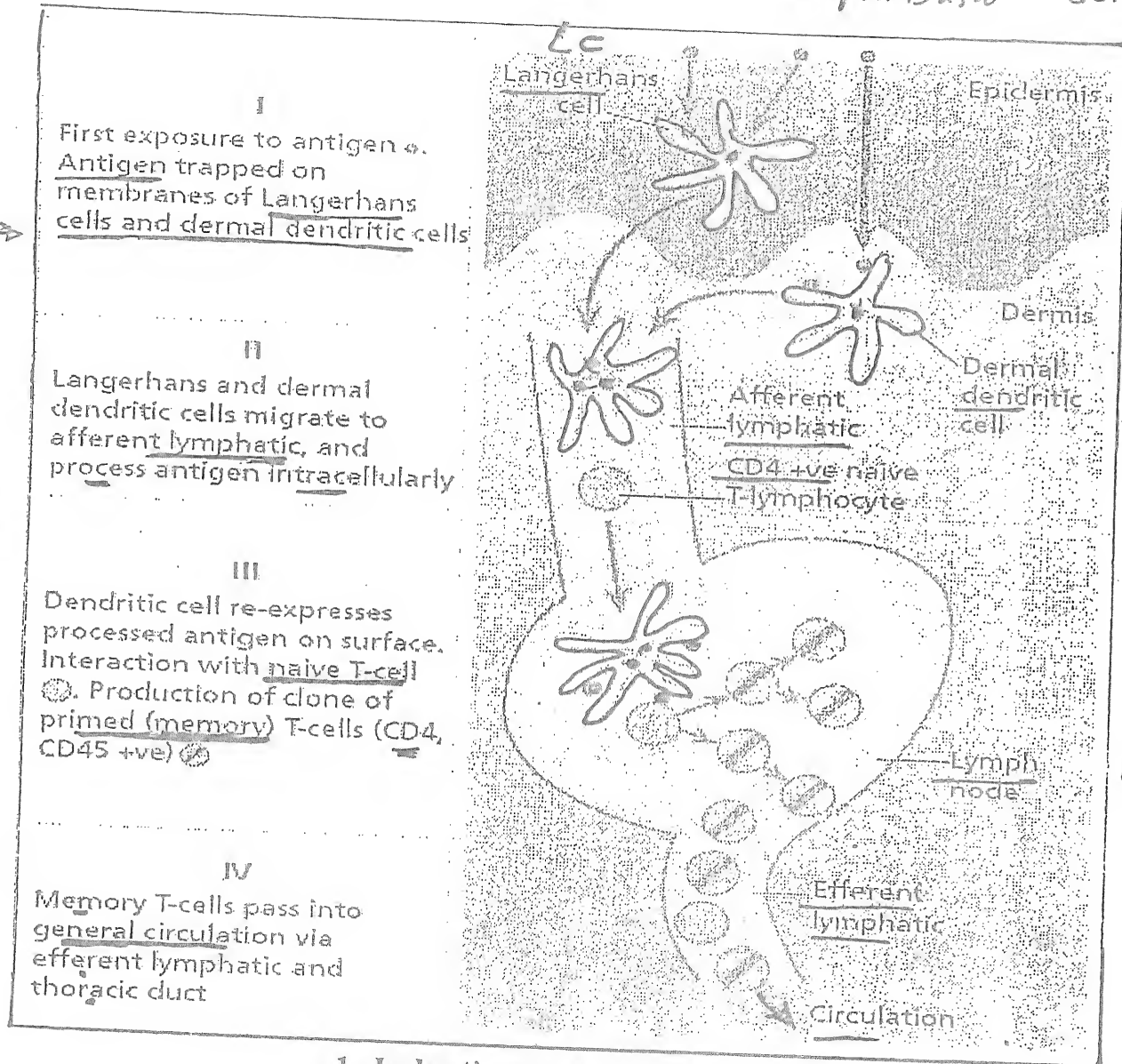
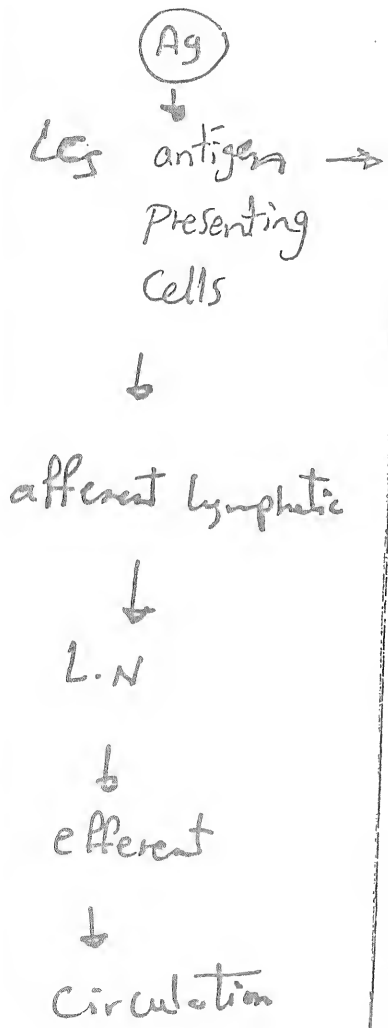
- Previous contact is Needed to induce Allergy.
- Specific to one chemicals & close relatives
- After Allergy has been Established all areas of skin will react to the Allergen

طول العمر

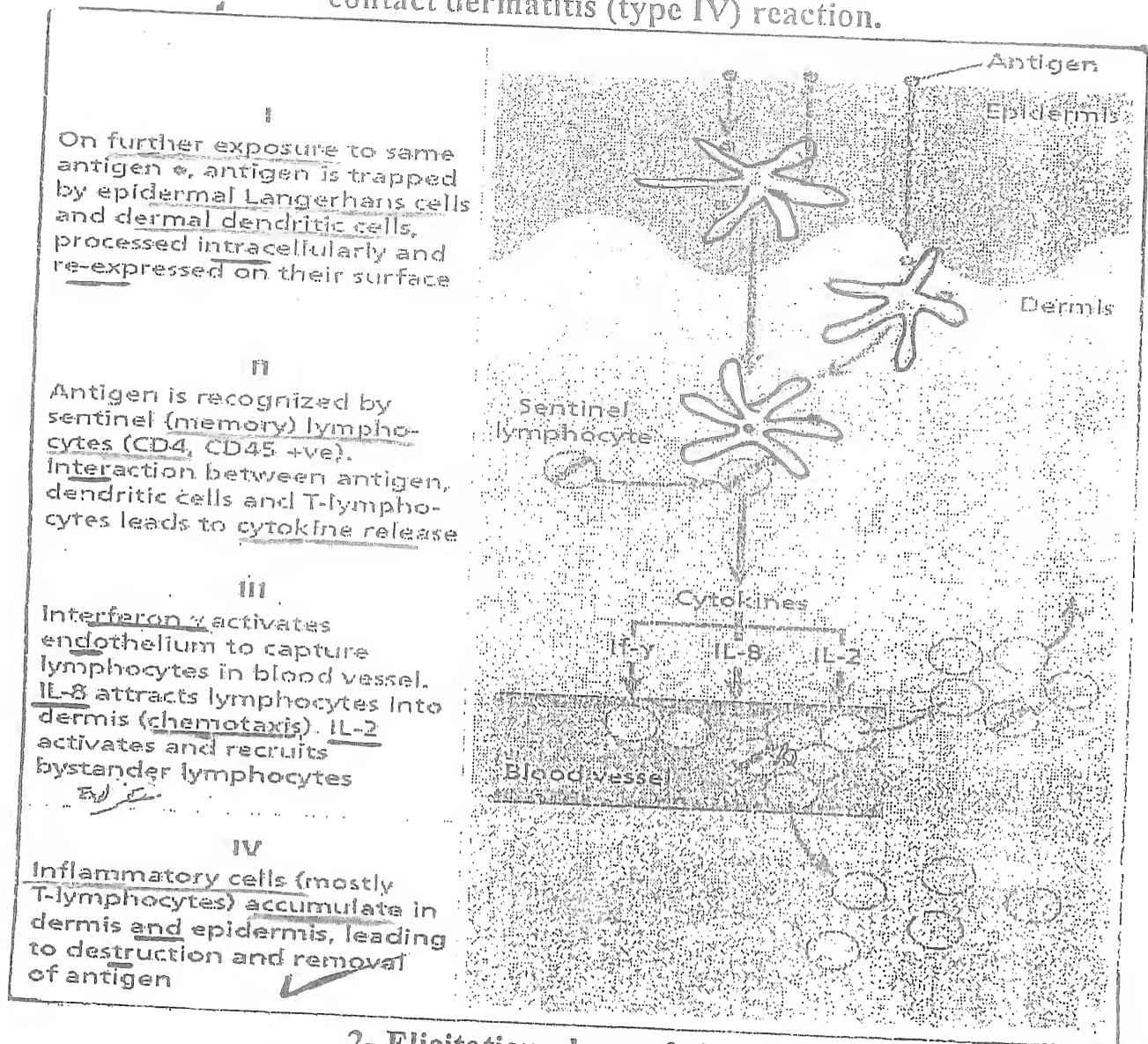
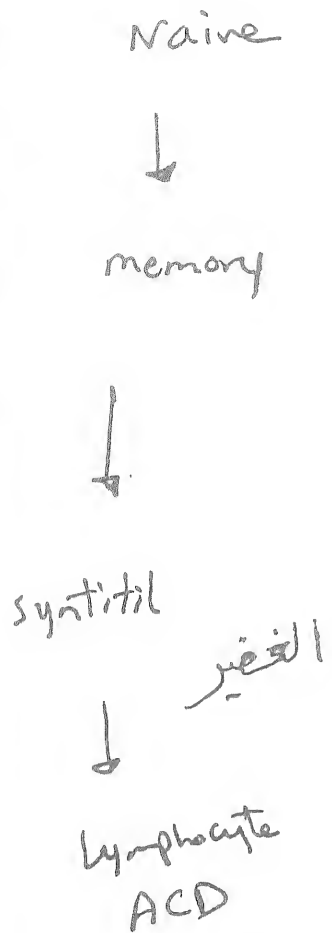
النزعة نادرة

- Sensitization persist "indefinitely"
- Desensitization ↓ - seldom occurs.

Suprabasal dendritic cells



1- Induction phase of allergic contact dermatitis (type IV) reaction.



2- Elicitation phase of allergic contact dermatitis (type IV) reaction.

ناب

IL8 → Chemotaxis
 IFN-γ → ++ Endoth. → Capture T_H B_Vs.
 IL2 → ++ recruit Bystander L

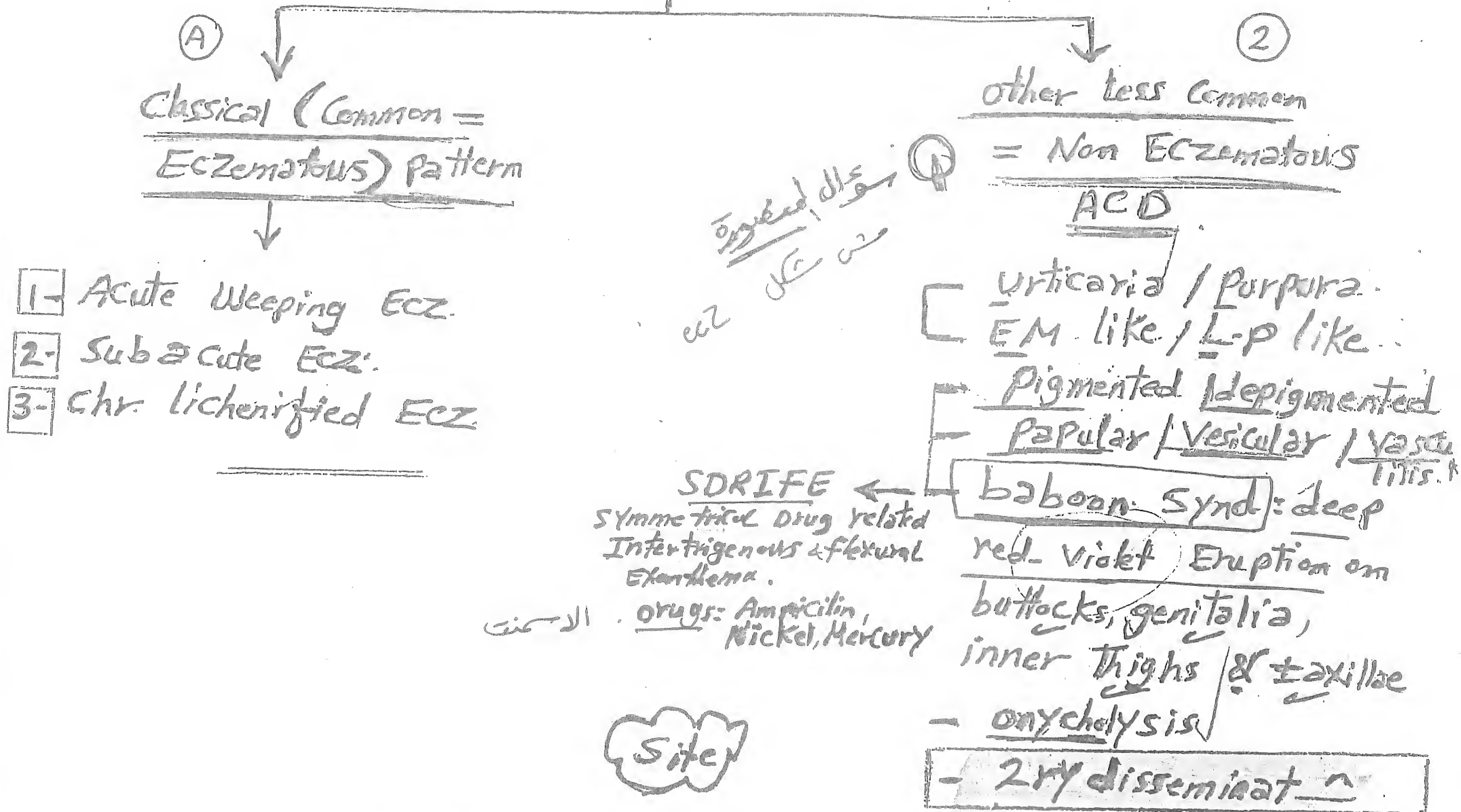
NB → IL8
 ACD
 ICD

ACD ← dermis // B.V.s no lymphocyte

Clinical picture of ACD :-

- A. Clinical presentation onset الداء
- B. Causative Allergens مقا
- C. Regional ACD الاح Ⓢ

A. Clinical presentation



usually localized to site of Allergen

Contact but Generalization may occur

(Morbilliform or Exfoliative or Dry disseminated) ACD.

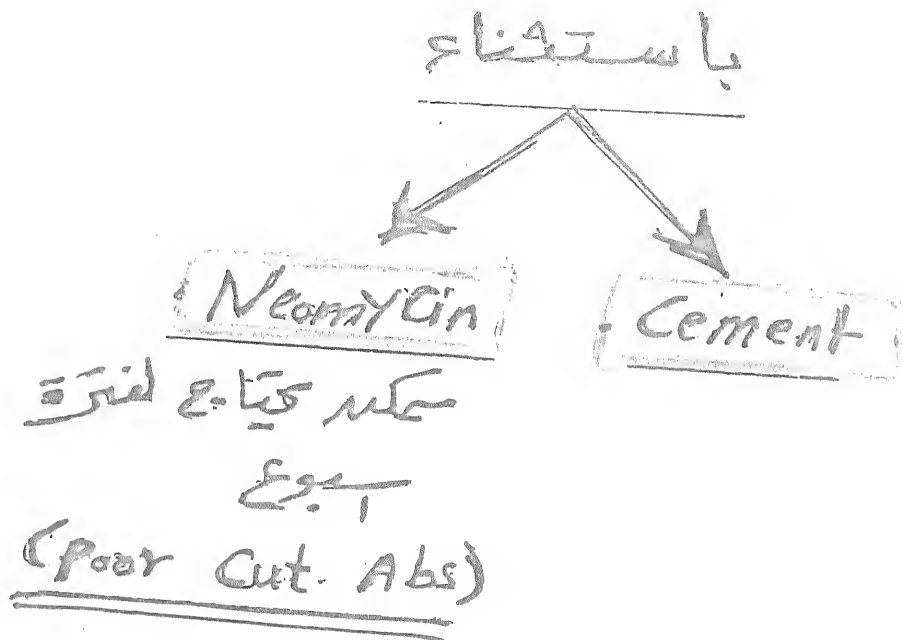
Rash
Ida
reaction

→ The initial site of dermatitis often provides the best clue regarding the potential Cause of ACD. (See regional ACD).

onset

usually 1-3 days from Contact & Specific

Allergen in patient who have previously sensitized by the same Allergen (= Elicit phase)



NB : Induction phase may be caused by a Topical agent while Elicitation phase caused by the same Agent but when given systemically → ACD w ±:

- ① localized to site of previous contact or
- ② 2ry disseminated. Generalized

* This Condition Called « **Systemic ACD** » eg Cinnamon oil & other medications.

B. Causative Allergens

→ About (25) chemicals responsible for 50% of ACD Cases → See the table.

≥ 3000 Ag

→ Commonest of them are: (10)

- Also : Toxicodendrons (Poison Ivy, oak, sumac) is a classic Example of ACD in North America w is ch-by linear streaks of ACD that develop when plant parts have been in direct contact w skin.

(usually: fingers, Wrist forearm, genitalia)



<p>ناتج امرتيا</p> <ul style="list-style-type: none"> Nickel. Poison IVY Fragrance Mix 	<p>ع دوي</p> <ul style="list-style-type: none"> Neomycin ✓ Bacitracin ✓ Topical Cs ✓ Thimerosal 	<p>ناتج</p> <ul style="list-style-type: none"> Gold chromate Cobalt 	<ul style="list-style-type: none"> Palson of para paraphenylenediamine formaldehyde Rubber
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Top 10 Allergens Identified by NACOG

Note

Metals (1, 6, 10)

Sensitizers (3, 7, 8)

Topical Antibiotics (2, 9)

Fragrances (3, 4)

① Nickel

② Neomycin

③ Balsam of Peru

④ Fragrance mix.

⑤ Thimerosal

⑥ Gold

⑦ Quaternium 15

⑧ Formaldehyde

⑨ Bacitracin

⑩ Cobalt

حاجات
Poison Ivy
Chromite
Rubber
Cs

Nickel

الأشهر
المجد - المعلب
المحفوقات

Commonest
in Female

*It and Poison Ivy are the commonest allergens at all (شفوي)

يمكن بيبقي موجود في:
① الأكل: الاسباجيتي المعلب - الفول المعلب - والخضروات والفواكه المعلبة -
بيسكويت - البيض - الشاي - الحبوب - الحمار - لوز - إسبانج - الشيكولاتة
② المعادن زري:

*الحلق (لو كان فالصو او ذهب بس الكليس نيكل).
earrings
*العقد (الرقبة)
neck lace
*زرار الجينز (jeans stud)
*خرامة الاذن
*العملات المعدنية (حساسية علي الجلد اللي تحت الجيب)
*سماعة اليد (مغصمها او الاطار)
hand watch

Commonest sites of nickel ACD

- *Ear lobes....from earrings or piercing by nickel material.
- *Neck....necklaces
- *Wrist....watch strap
- *Lower abdomen....jeans button or stud
- *Hand....pompholyx

لاحظ: عشان تمنع حساسية فتحة الاذن مكان الحلق:

١- استعمال خرامة اذن من الاستانليس وليس من النيكل.

٢- ممنوع استعمال الحلقان الفالصو

٣- لازم كليس الحلق الذهب يكون خالي من النيكل (ذهب او

قصدير).

ملحوظة: الاكلات التي لا يوجد بها نيكل:

السماك - اللبن - البيض - اللحم - البط - القهوة - الخضروات
والفواكه الطازجة

مفضل

Neomycin 7 Cs

Bacitracin

- Topical antibiotic used in skin, eye, ear and piles preparations, alone or with bacitracin, Cs, or antifungal eg. Kenacomb cream.
- Crossreaction with bacitracin, gentamycin, streptomycin may occur.

Balsam of Peru

التوابل
بالحففات

Naturally occurring fragrance used in: Some scented cosmetics, spices and suppositories.

تشوفها في المرضي اللي عندهم حساسية من الطماطم - الموالح -
القرفة - cinnamon - القرنفل - الثوم - الخمرة - السجائر.

Fragrance mix.

عطر - حذا
عبر

- Most common cause of allergy to cosmetics

- Uses: * To give pleasant odor (مكسبات رائحة) ←

* To mask unpleasant odor:

عشان كده بتبقى موجودة في المستحضرات المكتوب عليها
(unscented).

- So use fragrance free preparations

	NB: allergy to cosmetics may be due to: fragrance mix, preservatives (Q.15, dyes and lanoline).
<p>ثيمروزال</p> <p>حادة حافظة</p> <p>الحلول الدسات</p> <p>التطعيمات</p>	<p>Preservative material used in:</p> <p>1 Vaccines</p> <p>2 Contact solutions</p> <p>3 Ophthalmic and ID testing solutions</p> <p>4 Antiseptics and cosmetics</p> <p>(لو واحد بياخد مستحضر للعين او الاذن وحصلتله حساسية منه؟ ايه ممكن يكون السبب) (2)</p>
<p>Gold</p> <p>"الكثير لو تم استعماله"</p> <p>"في الاذن"</p>	<p>* Inert material used in jewelry, dentistry, electronics</p> <p>* Rarely considered as a cause of Jewellery dermatitis but this common if the patient is wearing dental gold</p> <p>* presentation: - hand, facial and eyelid dermatitis</p> <p>- Oral lichenoid eruption</p>
<p>Formaldehyde</p> <p>(colorless gas)</p>	<p>Colorless gas used in: Shampoo, cosmetics, newsprint, deodorant, smoke, car exhaust and clothing (يخلطها تشف) wash and wear antiwrinkles</p>
<p>Quatrineum 15</p> <p>مضاد حيوي - مضاد فطري</p>	<p>Preservative with antibacterial and antifungal properties.</p> <p>- used in shampoos, moisturizers, soaps and cosmetics (commonest preservative)</p>
<p>Cobalt</p> <p>المجوهرات</p> <p>الغنا</p> <p>الطوب</p> <p>صبغات الشعر</p> <p>السيراميك</p>	<p>معادن يخلط مع المعادن الاخرى لاعطائها الصلابة والقوة وخصوصا النيكل والكروميوم وده اللي بيخلي المريض الواحد دايما حساس للتلاتة معا.</p> <p>Uses: Jewelry, dental plates, prostheses, polish stripper, hair dyes, ceramic, enamel</p> <p>NB: Patch test to it may produce specific reaction called Poral which is erythematous violaceous dots not due to cobalt allergy but due to allergens residing in acrosyringia.</p>
Bacitracin	<p>As neomycin. Cross reaction with neomycin may occur.</p> <p>NB: rarely cause anaphylaxis or contact urticaria.</p>
<p>Corticosteroids</p> <p>type I, IV</p> <p>hypersensit</p>	<p>May cause ACD suspected by failure of response or worsening; may be + d.t (Cs) its d.t for d.t preservatives</p> <p>4 classes of Cs are known:</p> <p>A. Hydrocortisone and tixocortol group</p> <p>B. Triamcinolone acetinoid and budesonide group</p> <p>C. Betamethasone group</p> <p>D. Hydrocortisone 17 butyrate.</p> <p>Tixocortol and budesonide are best used as screening to ACD to Cs.</p>
<p>Paraphenylenediamine</p>	<p>الحنة - الصبغات</p>

Q C. Regional ACD

15
 والتب عن +
 Allergens
 كانه المهوره

Region	Possible Cause of ACD
Scalp	<p>relatively resistant to ACD but it may occur d.t.:</p> <div> <div> <p>hair dyes</p> <p>Shampoos</p> </div> <div> <p>Hair sprays</p> <p>Hair straighteners (الجلات)</p> </div> </div> <p>Scalp دائما مشين يحصل في scalp ولكن على الأذن والوجوه < + postauricular & Neck</p>
Face	<p>In general; CD of Face may be:</p> <ol style="list-style-type: none"> 1- ACD → Face Creams, Powders, shaving lotions 2- Airborne ACD → ACD involving sun exposed & protected areas (Eyelid - postauricular & below chin) → الربو الحبيبي 3- Photoallergic CD → as (2) but differentiated from it by sparing of sun protected Areas. <div> <p>صوب اللق + dust of cement</p> <p>مكانه مغفلة</p> </div> 4- Irritant CD → Subclinical ICD may be d.t. Some Cosmetics. <div> <div> <p>C/P</p> <p>burning, stinging, or Contact urticaria</p> </div> <div> <p>تشوفا</p> <p>كثير</p> <p>فهره</p> <p>التهاء</p> </div> </div> <p>DD ACD</p>
Ear	<p>Rim & Postauricular → دائما حاجة ان تطبق على scalp وجملت ACD (الجلات) - التهارة</p> <p>Lobule → Nickle ACD (earrings/piercing) (حلقة / فزمة)</p> <p>EA. Canal → استعمال نفق الأذن للعلاج تنوى على Thiminasal أو Neomy Cin or bacitracin</p>

fragrance mix

<p><u>Eye lid</u> (عين)</p> <p>unilat → (ACD) Bilat → (AD) Recurrent seborrheic</p>	<p>1. <u>Nail Polish</u>: Commonest site of Nail Polish ACD is Eye-lid dermatitis (unilat).</p> <p>2. <u>Mascara</u></p> <p>3. <u>Fragrances</u>.</p> <p>4. <u>False Eye Lash Adhesive</u>.</p>
<p><u>Lips & Perioral</u></p>	<p>• <u>Lips ACD</u>: ↳ Lipsticks ↳ Tooth paste ↳ Nail Polish.</p> <p>• <u>Perioral ACD</u>: ↓ Extension from Lip CD.</p> <p>• <u>Lips & peri-oral</u>: ↓ Citrus Fruits & Mango → phototoxic Lip & perioral Dermatitis.</p>
<p><u>Neck</u>: اللو</p>	<p>1. <u>Perfumes</u> → ACD or phototoxic CD.</p> <p>2. <u>Nickel</u> → of Clasps of Necklaces (سلسلة)</p> <p>3. <u>Textiles</u> → Collar like dermatitis or "Neck side dermatitis" (عرق-سيرة)</p> <p>4. <u>Air borne ACD</u>: So ACD in this case is sharply limited by collars.</p>
<p><u>Trunk</u></p>	<p>→ <u>Uncommon</u> site of ACD</p> <p>• <u>Axillary ACD</u> may be d.t.</p> <p> ① Axillary Vault → deodorant (Apex)</p> <p> ② Axillary Folds → clothing dye.</p> <p>• <u>Brassieres</u> may cause ACD from:- ✓ Material itself ✓ Elastic & metal <u>snaps</u>.</p>
<p><u>Abdomen</u></p>	<p>1. <u>Waist line</u>: "Rubber dermatitis" from Elastic in pants & undergarments. (سلسلة)</p> <p>2. <u>periumbilical</u>: Nickel in Jeans Button or Stud.</p>

الموالح

Genion

1. Genion → usually spared.

2. buttocks & upper thighs: clothing dyes

3. Penis: • Poison Ivy

• Condom ✓

• Transmitted by fingers (علاج كدمات)
ACD

4. Vulvae: (50%) of pruritus Vulvae is due to ACD
From: medicaments, fragrances & preservatives

5. Perianal: • Mediators: Caine
Neomycin
• Cleansers: contain preservatives

Upper limb

Wrist Nickel:
Chromes leather wrist-bands.

Hands:

Site ±
Dorsum
Palms
Ring site
Web spaces
Finger tips

Allergens
Fluorists
Nickel, chrom
Rubber
Poison Ivy

Pattern: may be Pompholyx like (لويحات)
Nummular streaks.

L.L

1. Shins: "Rubber" From Elastic stockings

2. Feet: v. Rubber

(shoe dermatitis) • chrome tanned leather
• dyes
• Adhesives

Antibiotics & moisturizers applied to stasis D.

أستد →

Location	Suspicious Agent
Eyelids	Nail polish, eye makeup, airborne allergens
Eartlobes or neck	Metal jewelry
Forehead, scalp margins	Hair dyes
Face	Cosmetic fragrances and preservatives, airborne allergens
Axilla	Deodorants
Hands	Gloves, occupational contacts
Waistband	Elastic
Dorsal feet	Shoes

• Invs. of ACD

(A) Patch Test

(B) Photopatch test

(C) ROAT test

(D) Dimethyl-gloxime test

(E) Histopathology

↓
Sec
(ECZ.)

(A) - Patch test "Type"

• def.

$\frac{1}{2}h$

24-48h

• Types

7-21d

• Methodology

• Results.

def.: Special test used to detect Hypersensitivity to a specific substance (Allergen) that is in contact with the skin so that the allergen may be determined & corrective measures taken.

Types

TRUE test [standard screening Test]

(thin Layer Rapid use Epicutaneous test)

بازر

Finn Chambers test

(Individually prepared Aluminium chamber mounted on Scanpor Tape)

بازر

prepackaged Allergens

No of Allergens (23) (there is one control w accounts for the 24th spot) → نازلہ

لہذا (Ag 23) +
مقابلہ ملی نازلہ
تکے رکھو تاکہ
لو سے پاک رہے



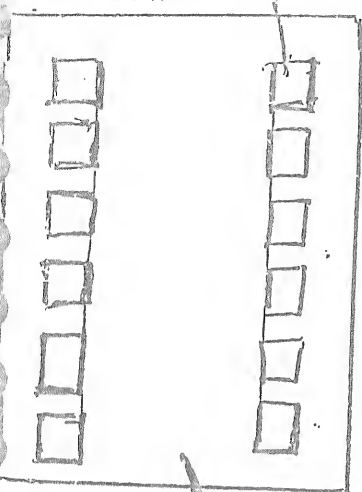
— Allergens come in multi-System Syringe & placed into Finn chambers mounted on Scanpor Tape

— allows Testing for (50+) Allergens (Expanded Series of Allergens

➤ TRUE Test).

معنى باختصار

مربعات
تحتوي على
(Allergens)



TRUE test :

مريض بأكبر كيار كل واحد يحتوي على
12 مربع صغير يتخلوا على Allergens
المواد الكشف عنه، خاصة لديهم
وعلى شانه يمشري جاهز فهو هل الاستحذام
ولكن عدد المواد (Allergens) الموجودة فيه صغير (٢٣)
(pre packed Allergens)

Finn Chambers Test :-

لبلاستر كبير
يوضع على الظهر

يتم اختيار Allergens المواد اختياريها
(لانه قابل)
في سرجات ثم يتم وضعها في حجرات من البلاستيك (Scan por tape)
مثبتة على بلاستر وتوضع على الظهر
يتم اختيار عدد كبير من Allergens أكثر من TRUE test
(Expanded Series of Allergens ≥ 50)

NACDG
Series



European Standard
Series

وعالبا كل بلاستر يبين ملازق عليه حوالي ١٢ حجرة تحتوي على ١٢ Allergen

(50) if TRUE Test is

- ve

Finn Chamber test

(معنى لعال ٢٣) مادة طلعوا (ليست) من مواد
أخرى إيجابية في Finn test

احضره

Methodology:-

- ① Ask the patient To bring all his own materials for testing:-

يتخلل المريض يجب كل المواد التي يتعامل معها في البيت أو المستشفى
علائق تعرف مكوناتها وتكشف عدم Allergen الحساسية

ابقى المعد قليل
بأسبوع

→ Bring or send all chemicals for Testing (1 w) before the 1st appointment

→ only small amounts required (few drops or grains)

مشروع
المنظرة

then these products are classified as:-

leave on products as:

Moisturizers
[Make up]

↓
Tested as it
(من غير تخفيف)

rinsed off products as:

Soaps
[Shampoos]

↓
Tested after
dilution

ويجب ما تعرف مادة لفعالة في كل منتج يتعامل معه المريض
بتحققه بـ Petrolatum (بتكره صيدلية) (بجاء كل مادة)

وكدة يبقى حضرنا المادة المراد اختبارها
وتم وضعها في Finn chambers

احتياطات
قبل وبعد

② Precautions:-

قبل ما نلزمه بـ Patch

Avoid back Exposure for Sun 4 wks before Testing
Avoid CS at least 1-2 wks before testing
(>15 mg prednis)

بعد ما نلزمه بـ بلاستر

(No) Swimming, bath or Exercise → كدة بلاستر
ننقله

Arrange for someone to remark the test sites with "indelible" felt tip Marker

ارسم حول علامة
في مكان ما بتكره صيدلية

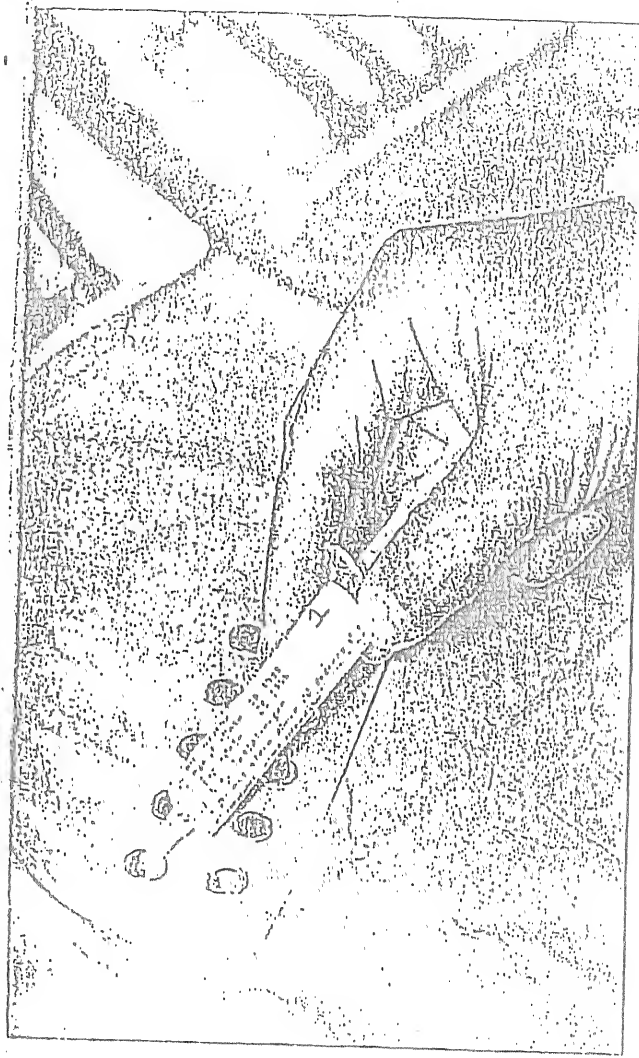


Fig. 15.11 Allergens contained within syringes being placed by nurse into Finn chambers.

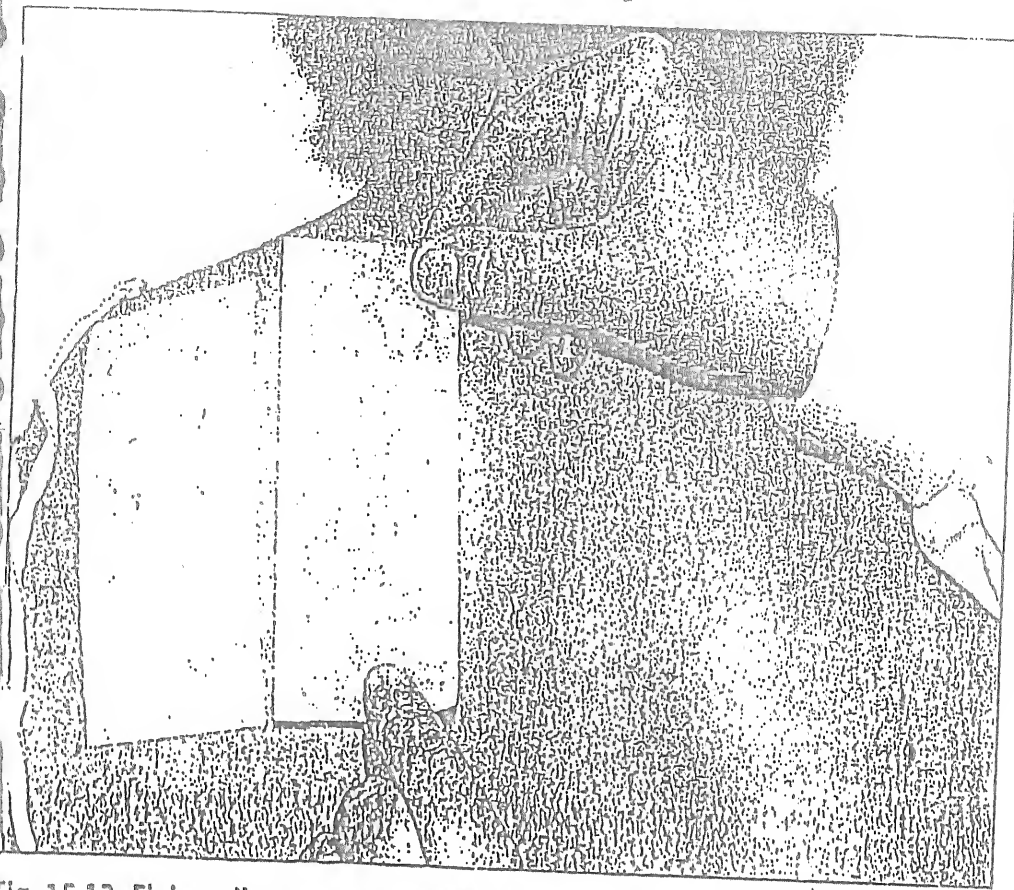


Fig. 15.12 Fixing allergens to patient's back using Scanpor® tape.

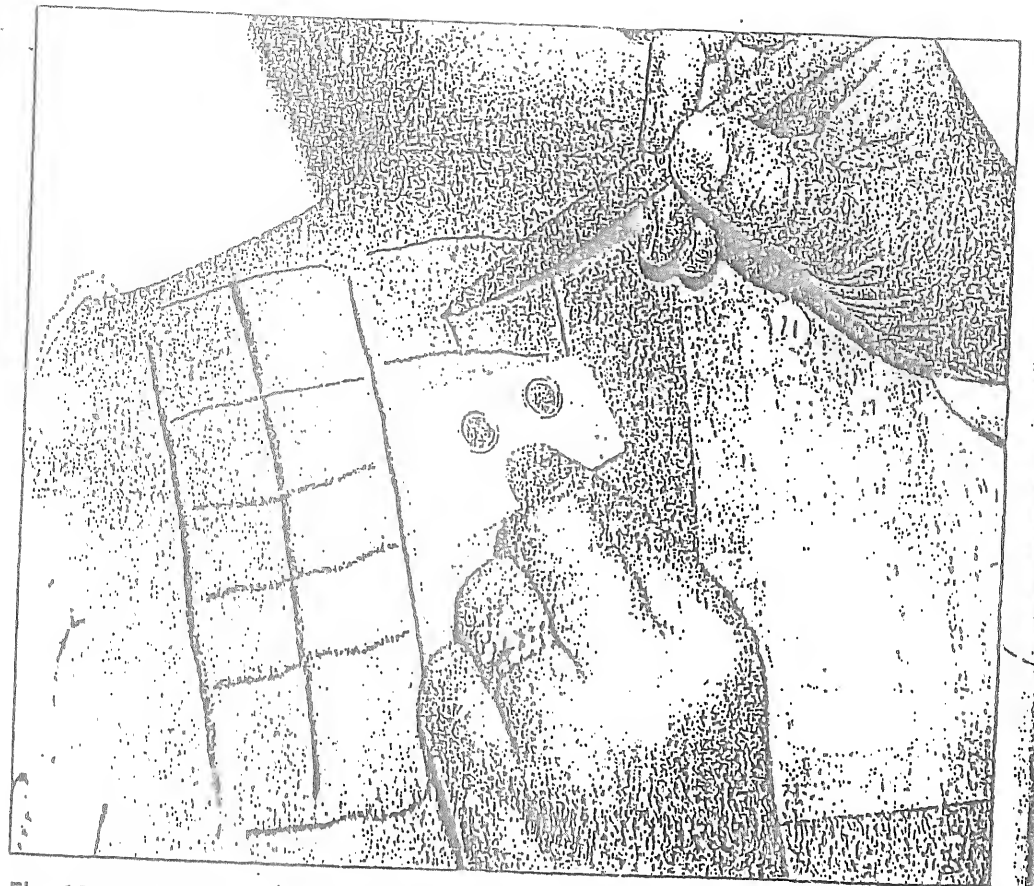


Fig. 15.14 Allergens being marked upon removal of Scanpor® tape.

3 - Reading:

- The patient returns after 2 ds (48 hrs)

متيأ كورس ان ليلاستر لسه لانتق
كوديس (جرات Finn بيتي عامله علامات
على الجلد)

يتأخذ قرائنين؟

① Patch is removed & Readings are taken:-

② Readings taken at:

①st reading → 48 hrs after application (عريفه - اميا -)

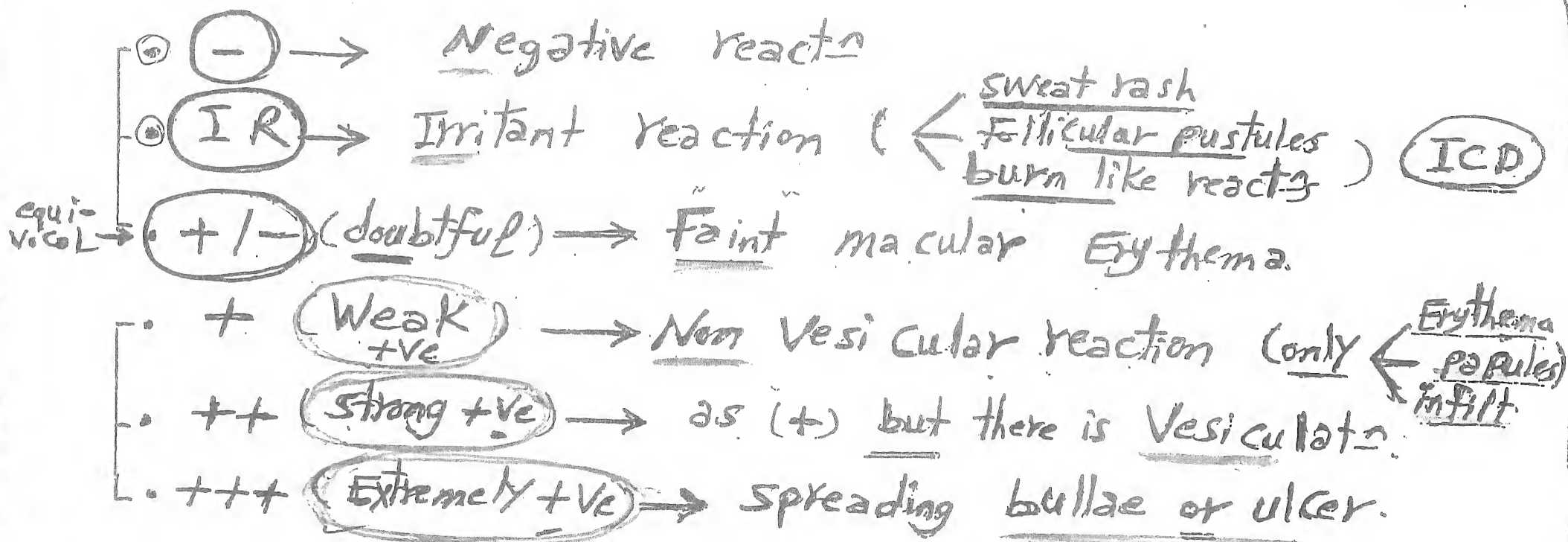
②nd reading → 3 - 7 ds (after appln) Why

→ Some allergens as Gold & Neomycin dyes may be delayed.

النتيجة

Results:-

(According to International Grading System for patch test)



note Q

NB: Angry back Syndrome = Excited skin synd.

Def.: state of Hyper excitability ch By False +ve reactions (The -ve Tests appears weakly +ve).
It may be d.t:

AET: ① Active dermatitis at time of patch testing or

hypoallergenic
Plaster
البلاستر
تقوية

② strong +ve reaction → induce local skin hyperreactivity in areas where patches are applied.

Management: re-testing the patient sequentially to small series of these Allergens.

لو كان ليس
رقم
لو كان ليس
رقم

→ # of Active Dermatitis.

B - photopatch test = (for D of ...)

This test used for evaluation of photoallergic CD

To such substances as: - sulfonamides
- phenothiazines
- PABA → in Sun Blocks
- oxybenzone

احذر
على مكانين

these: Chemicals are applied in duplicate sets.

استخدم

one set: receives 10 J/cm² (or 1 J/cm² less than MED to UVA).

other set: the other series protected from UVA

الشخص
(التي هي)

(i). +ve React on Both sides → ACD

(ii). +ve " at UVA exposed side only → PAGE

C-ROAT Test

اجربا على
ايضا مثالا

(Repeat Open Application Test)

Indications: (1) -ve patch test despite there is strong suspicion of ACD.

(2) Weak +ve patch test (specially for leave on consumer products)

Ref.
Secrets

(remember that: Patch test is one time occlusive test that doesn't duplicate low level chr. daily Exposure)

← هاجيب المادة دي (لو جوده مثالا في مطبخ) ويتم استعمالها مرتين
ليومين على جانب ارقية - قلبك يذنب - أو على الذراع لمساحة (مربع) اكم
لمرسل ← ACD ← "the weak patch test is highly Relevant"

D. Dimethylglyoxime test

def useful & practical way to Identify Metallic objects that contain enough Nickel to provoke ACD to Nickel.

① ممكن دكتور الجلدية يعمله في الحياة أو المريض بيستخدم Kits ويعمله في البيت.

② توضع نقطة بسيطة من هذه المادة على (قشرة) أو شاشه ويتم تدليكها بالاشياء المراد الكشف
عن النيكل فيها. في مثال نوان لو اتحول لونه لاشاحق في الأحمر ← +ve For Nickel

• Treatment of ACD

✓ ① Remove the Allergen xx

✓ ② Treat the Eczema كالمعتاد

ملاحظة

Irritant CD

(ICD)

def. Cutaneous inflammatory disorder resulting from direct cytotoxic effect of chemical or physical Agent. (Non Immunologically mediated).

Epidemiology :-

- Represent (80%) of cases of CD
- Represent (80%) of occupational skin disorders

Pathogenesis : → 3 main pathophysiological changes :

- Cytotoxic + 2D → disrupt denat.
- (i). Skin barrier disruption → ↑ TEWL
 - (ii). Epidermal cellular changes.
e.g. Keratin denaturation.
 - (iii). Cytotoxicity: release of $\left\{ \begin{matrix} \text{TNF}\alpha \\ \text{IL6} \\ \text{IL1B} \end{matrix} \right\} \rightarrow \text{cytotoxicity}$

ICD has 2 phases: (HL)

↓
Acute

- ① Penetration by the irritant Agent produces 3 changes (تغيرات)

The Most important cytokines are:-

- TNF α
 - IL6
 - IL1B
 - IL8
- ↑ upregulate Expression of ICAM-1

↓
Chronic

↓
damage to Stratum Corneum lipids (mediate St. Corneum barrier function)

- ① loss of Kcs cohesion → desquamation
- ② ↑ TEWL

Causes of ICD:-

- Almost any material can cause ICD if there is sufficient Exposure both in
- ① Time & Concentration. ②

→ Most Common irritants are:

IRRITANTS AND MECHANISMS OF TOXICITY	
Irritant	Mechanisms of toxicity
Detergents	Solubilization and/or disruption of barrier lipids and natural moisturizing factors in the stratum corneum Protein denaturation Membrane toxicity
Acids	Protein denaturation Cytotoxicity
Alkalis	Barrier lipid denaturation Cytotoxicity through cellular swelling
Oils	Disorganization of barrier lipids
Organic solvents	Solubilization of membrane lipids Membrane toxicity
Oxidants	Cytotoxicity
Reducing agents	Keratolysis
Water	If barrier is disrupted, cytotoxicity through swelling of viable epidermal cells

إنتانات مطهرات

Hydrofluoric
Sulfuric acid

NaOH
في ملامسة الجلد
وأضرار الجفاف

ليترين وغازولين

Acids
Alkalies
Detergents
Disinfectants
Solvents

Plastics
Fabrics
Water

Bodily fluids:
Saline
Urine
Feces
diaper D.
lip licking
Fecarnet III
→ Penetration
ICD
Food & its additive

Cement

الأسمنت يمكن جعل شغري

① ACD: d.t Chromate

② ICD: d.t its Alkalinity

2 Types:

Acute delayed

بجمل بعد ١-٢

Cumulative

بجمل بعد

١-٢

من التعرض

المستعمل

المريض يستغرق
طول عمره شغل
في الأسمنت ولباسه
دعا محله

3. Both: ACD & ICD (ACD may accompany Cumulative ICD)

• CLP of ICD

As General picture of

Dermatitis (or) ECZEMA = it may be:

• Eczematous

• Non Eczematous

- ✓ Burning Sensation
- Chemical Burn like.

- Pustular ✓
- Aciciform ✓
- folliculitis ✓
- Xerotic ✓

① Acute →

② Subacute →

③ Chronic →

Types of ICD:

1- Acute Immediate ICD (minutes-Hours)

حساسية فورية

Strong irritant
short period

2- Acute delayed ICD: (8 - 24 hrs)

حساسية متأخرة

e.g anthralin

3- Cumulative ICD: (chr. ICD)

[weak irritants
Repeated Application

متراكم

occurs as a consequence of multiple subthreshold skin insults without sufficient time bet. the m for complete barrier function repair.

- Acute Immediate
- Acute delayed
- Chr. Cumulative
- Irritant React- ICD
- Pustular & Aciciform
- non Erythematous (Subclinical)
- Subjective or Sens.
- Atopic
- Traumatic
- Frictional
- Air borne

then
 → Cumulative ICD develops (e) subsequent exposures (or) when threshold of irritants is ↓↓ e.g AD.

(4) Pustular or Acneiform [Folliculitis]

e.g mineral oils, tar

بالقوة
 (التهيج)

(5) Non Erythematous ICD: Pathological ICD

(6) Subjective (Sensory):

(Not) Clinical

sub-clinical

only stinging / burning sensation

(7) Air-borne ICD:

(8) Frictional ICD: due to repeated frictional Trauma

(9) Traumatic ICD: Irritant is applied over sites of Trauma e.g

Burns & Lacerations

(10) Asteatotic ICD: [See Xerotic ECZ.] ✓

(11) irritant reaction ICD: subclinical (12) wet chemical environment (hair dresser)

التهيج الحاد

Sharp outline

extend beyond site

TABLE 2-1: COMPARISON OF IRRITANT AND ALLERGIC CONTACT DERMATITIS		
	Irritant (8%)	Allergic (2%)
Examples	Water, soap	Nickel, fragrance, hair dye
Number of compounds	Many	Fewer
Distribution of reaction	Localized	May spread beyond area of maximal contact and become generalized ✓
Concentration of agent needed to elicit reaction	High	Can be minute ✓
Time course	Immediate to late	Sensitization in 2 weeks; elicitation takes 24-72 h. ✓
Immunology	Nonspecific ✓	Specific type IV delayed hypersensitivity reaction
Diagnostic test	None	Patch test + ✓
IL 4	-	+
Incid:	More Common	less Common
CLP:	stinging or Burning	Itching
prognoses	Better	Bad →

Agents
 No Conc.

Mechanism

Incid
 Mech.
 Agents

CLP
 Diagnosis

Painful

Diagnosis of ICD: No specific test; diagnosis of exclusion; when Dermatitis can't be explained by -ve patch test to a known allergen.

Photo Contact Dermatitis.

د. د. د. د. د. DNNZ

def. Allergic or Toxic reactions occurs When certain chemicals are applied to the skin & subsequently Exposed to sun.

الآثار و ظهورها
لها على الجلد
(Topical Agent + UVR → photoContact dermatitis)
(photosensitizers)

Pathogenesis : see drug induced photosensitivity (Exogenous photoreactions or dermatoses).

Examples :-

1- Sunscreens :-

Salicylates
PABA

Benzophenones
Musk

2- Fragrances :-

① Musk ②

3- Others :-

SFU
Tar
Psoralens
sulfa

→ Cadmium

Topical Agent
(photosensitizer)

+

UVA
(320-400 nm)

also UVB & Visible Light (any light length 280-600 nm)

PhotoContact Dermatitis

1-3 ds min-hrs

Photo. ACD

ECZematous Eruption

(No Post-Hyperpigment)

(on areas Where the Drug applied & UVR Exposure)

Photo. ICD (Phototoxic CD)

Sunburn like Eruption

PIH (with Post Hyperpigment)

لونها فاتح

شاق
للحرق بين نوعين
Sec light & skin

Photocontact D:

photoAll. عاليا نوعا

Photosensitizers

Topical → Photoallergic CD

Systemic → Photoallergic & phototoxic

لها على الجلد

See photoreact

NB Examples of photoxic dermatitis:-

① Phyto photo dermatitis:

- Contact \bar{e} plants containing phototoxic substances + UVR \rightarrow Reaction
- \rightarrow CIP: streaky or bizarre pattern ✓
- \rightarrow The most common plants are those of Umbelliferae family (Parsnip, Celery, Parsley, Hogweed)

Better classified as Phytophoto rather than photo.

② Berloque (Berlock) Dermatitis:-

(Berlock = ThinKet or charm)

def: phototoxic reaction dt Contact with plants containing Oil of Bergamot + UVA Exposure.

the photoactive component is:-

Bergapten (or 5MOP)

Source:

- Bergamot lime plant
- Perfumes
- Fragrances
- Cosmetics (لبنان)
- Toiletries
- After shave lot
- Colognes
- Sunscreens
- Moisturizers
- Soap
- Detergents
- Air Freshness

Oil of Bergamot (80-120 mins contact to skin)

Mid-late summer
الوقت بين الصيف
البحر قويه
5MOP قوي
وقت الحش

"Citrus Hood"

+

UVA

Berloque Dermatitis

CIP \rightarrow 2 phases:
 \rightarrow Acute inflammation
 \rightarrow Hyper-pigmentation

سأذكر

1- Initial acute inflammatory phase: after 24hrs

Pattern g.
lesions.
Pendant
like

- Erythema
 - Edema
 - Vesiculatⁿ
 - Pain. (not pruritic)
- Bizarre Config.

or drop like

2- 2nd phase: → Hyperpigmentatⁿ (Chief Complaint after 1-2 wks.)

site: any site where perfume is applied & UVA Exposure. e.g. → sides of Neck, arms, Trunk.

NB: - usually → No acute inflammatory phase & the presentatⁿ is only at Hyperpigm.

duration of: Acute phase → 5-10 wks

Hyperpigm. II → 1-2 yrs.

Invs ① photopatch test (see photosensitivity)

② pathology. (HIP)

Treatment: -

Emollient
Bleaching cr

1- Avoid all preparations containing oil of Bergamot

2. Acute phase → Wet Compresses + Antiseptic lot + Analgesics + Sunscreen

3. Hyperpigmentatⁿ (self limiting but after long period)

Hydroquinone

Tretinoin

Dexameth.

Ethanol

Propylene glycol

- Sunscreen

- Hydroquinone 2% (1x2)

- Kligman's formula (??) → Low pH

→ Ellagic acid: Naturally existing polyphenol That -- Tyrosinase by copper chelatⁿ

2126

Exogenous ECZema

Dermatophytide → see fungal inf.

ECZematous PMLE → see PMLE.

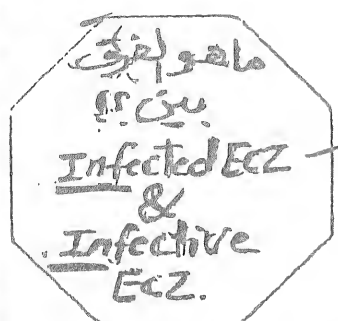
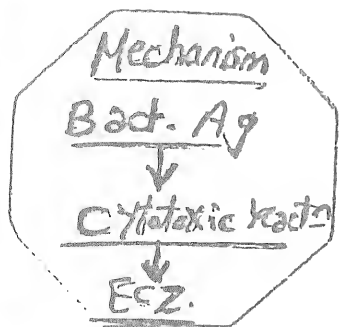
Infective Eczema
(infective eczematoid dermatitis, microbial eczema)

→ "caused by infection"

def: ECZ. Caused by contact with Microorganisms or Their products & cleared by eradication of the organism.

CIP: - Seen (few cms) from any site of inf.

- Can be seen in:-



ECZ is super
infection
over it

ما هو الفرق بين
Infected ECZ &
Infective ECZ.

1- Flexures (in scabies)

2- Scalp & postauricular areas (in pediculosis & impetigo).

3- around discharging sinuses, wounds & ulcers.

4- Around Molluscum lesions (Molluscum dermatitis)

5- Complicating T. pedis (dot (G-Vc) bact.)

6- Microbial feet ECZ: Weeping & Crusted ECZ

Seen on the dorso-medial aspects of Toes in persons with bad Hygiene or T. pedis.

7- infective dermatitis in children is HTLV-1.

- Severe Type of ECZ. Seen in Jamaican children in assoc. with HTLV-1 usually Generalized & Severe (Scalp, Postauric, Eyelid, Nose Perianal).

- may be an important marker for HTLV-1.

III → Systemic antibiotics Preferred as Topical ones may → CD.

Syst AB

Atopic Dermatitis (AD)

def of Atopy: a = No or without
 Topy = Topos = place } → out of place = غريب
 strange = unusual.

معناها → Atopy is an inherited tendency to develop استعداد
 a hypersensitivity response (Allergy) to certain
 Environmental stimuli (Allergens) is presence
 of chic Immunological profile.

Atopic dermatitis (AD): -

→ Cut. manifestations of atopic diathesis,
 Ch By chr. relapsing, intensely pruritic.
 dermatitis e chic distribution pattern
 in Atopic individuals.

* Atopic disorders (or) diatheses are:-

- ✓ Asthma.
- ✓ AD
- ✓ Hay fever (Allergic Rhinitis)
- ✓ Allergic Conjunctivitis
- ✓ Allergic Gastroenteritis.

" Etiopathogenesis "

- ① Genetic Factors
- ② Immunological Factors.
- ③ External Factors = triggers
- ④ others

- Hygiene theory

- Abnormalities of sweating
- Change in cutaneous lipids
- ↓↓ pruritus threshold
- Change in PDE activity
- Altered vascular and neurocutaneous reactivity

I. Genetic Theory (Factor)

in AD:-

- Autosomal dominant Inheritance (AD) ✓
- If 2 parents are affected by AD: → (80%) Incid. of Sibling Affected
- If 1 parent is " by AD: (20-50%) (60%) Incid.

Xerosis
 ± ASS
 Ichthyosis
 Vulgaris.

الخشونة
 غالبا

Defective Filaggrin Gene (FGL)

(20-50%)

(60%)

Incid

chromosome
 long arm
 segment
 19/23 →

Defective barrier →

↑ TEWL

Trans epid
 water loss

II. other Factors

والذين لديهم ما يسمى
 بـ "التهيج الجلدي"
 - النقرس
 - الحكة

① Hygiene theory: ↑ Exposure to Infectious Agents

during early life → (↑ Th1) to be potent

Th2 → ↓ Incid. of AD.

العرق

② Abnormalities of Sweating: in Atopics → ↑ Sensitivity

of Glands to Catecholamines → ↑ Sweating

→ taken up by st. Corneum → gland obst.

→ Sweat Retention → gland Rupture → diffuse to dermis → Itching.

cut lipid

③ Abnormalities in cut. lipids:

↓ Glycerol, Ceramide & Linoleic
 ↑ Epid. lipids.

→ All cause Xerosis.

الحكة

④ ↓ pruritus threshold: Early & prolonged experience of itching

⑤ Altered Vascular & Neuro cut. Reactivity:-

VC

" Paradoxical VC "

↓

يقل

So Atopics have Pallid skin &
 White Dermatographism.

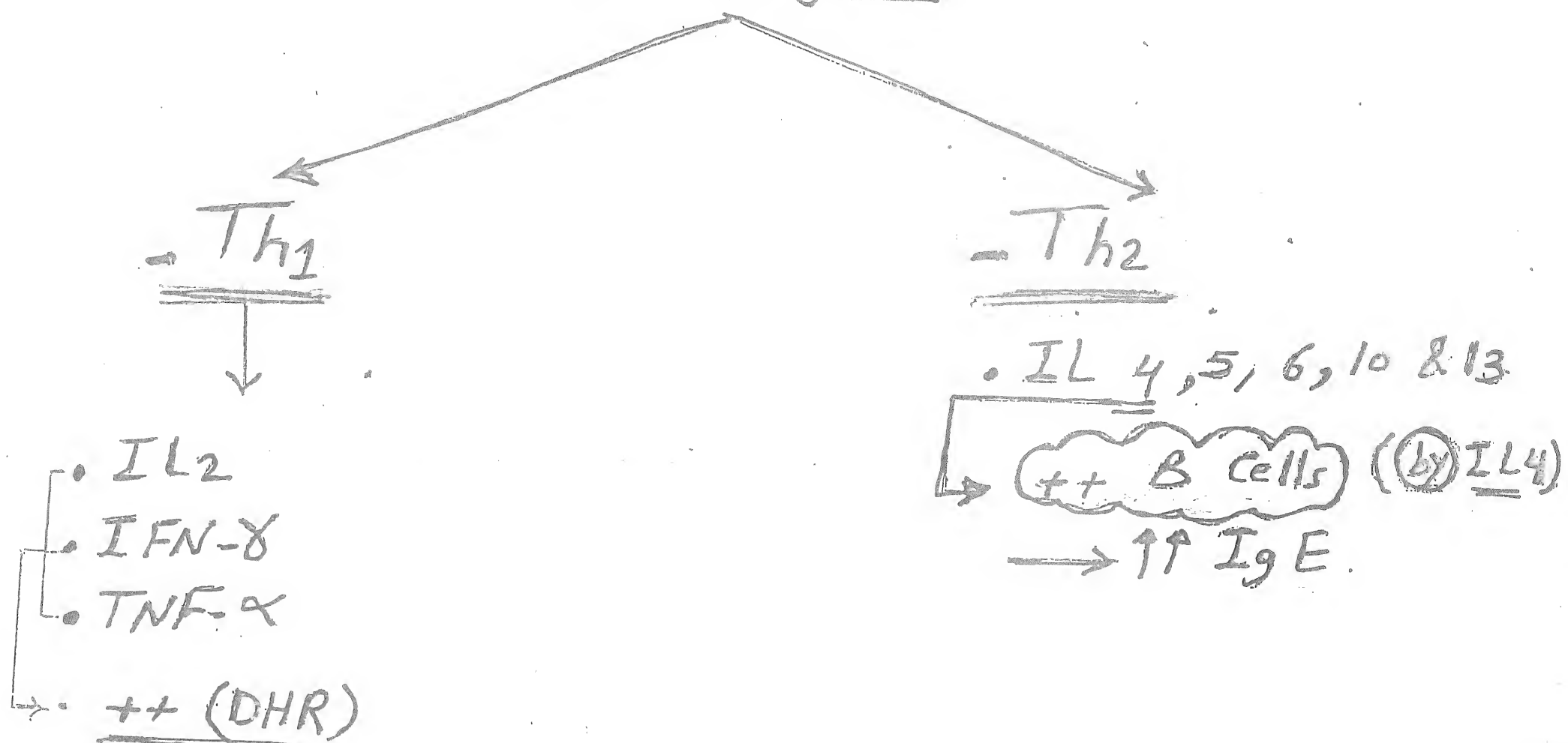
II Immunological factors:

T-cells
Igs
LCS
Eosinophils

12

T-Lymphocytes

CD4 or T-helper cells are of
2 subtypes:-



In NL individuals: there is balance between

Th1 & Th2 → each exert inhibitory action to the other.

In Atopics: → Th2 predominates in Acute & subacute phases while Th1 predominates in chronic phase.

So:

The Immunological Profile in Patients of AD is

Biphasic :-

Th2 predominate in acute & subacute lesions (Early phases)

1. \uparrow IL 4, 5, 6, 10, 13
2. \uparrow IgE
3. \downarrow IL2, TNF- α , IFN- γ
4. -- Type IV Hyper-

Sensitivity Reactⁿ (\downarrow Th1)

\downarrow Eliciting allergic (ACD)

\uparrow susceptibility for fungal & viral inf.

Th1 predominate in chr. lesions (Late phase)

- ① \uparrow IFN- γ , IL2, TNF α
- ② Reversal of Type IV (delayed hypersensitivity reactⁿ suppression)

IL4 level correlate late & dis. activity

NB: \uparrow IgE level \rightarrow \uparrow susceptibility to IgE Mediated diseases: e.g Anaphylaxis, urticaria, Atopy.

Immunoglobulins

- ① \uparrow IgE production.
- ② \uparrow IgG1 (ass. e air born allergen Hypersens)
- ③ \uparrow IgE4 (ass e Food Hypersensitivity).

NB \rightarrow \uparrow IgE \rightarrow not always a constant finding (see intrinsic AD)

LCS (Langerhans cells)

- \uparrow No
- \uparrow Reactivity
- \uparrow Expression of "FcER1" of IgE.

e.g. Wool

Eosinophils

\rightarrow Peripheral Eosinophilia / Hyperreleasable in response to non Allergens

$\uparrow \uparrow$

III) Extrinsic factors

① Irritants & Contacts:
 ← Solvents
 ← disinfectants
 ← Latex
 ← Nickel

② Airborne Allergens:
 ← House dust mites.
 ← Pollens
 ← moulds
 ← Mites.

③ Food (Diet):
 ← Milk
 ← Soya products
 ← Eggs
 ← Nuts

لبنة
موز
بيضة
البندق واللوز

علاقة الأكل بـ AD تتكهن في الآتي؟؟

① تؤثر على مجموعة معينة من المرضى، ومشورها بالحالات الشديدة.

② الأشخاص الذين ثبتت حساسيتهم من الأكل هم الذين

يؤثرون من وقت هذه الأمثلة.

③ الحساسية لبعض الأغذية وتأثيرها على [تأثير حاد ومعتدل

[على البنية الأولى فقط].

So food plays a minor Role

④ Microorganisms: ② Common organisms may act as a superantigens → Exacerbate of AD:

→ Staph. aureus ✓
 → Malassezia ✓

Severe AD of Face → Lotriderm or Pivarsone cr.

⑤ in head & Neck Dermatitis Ige to M. furfur can be demonstrated → Good Response to Antimycotic

⑤ Hormones
 ← Preg.
 ← menopause
 ← Menses

⑥ Exacerbate AD.

⑥ Stress: ↑ AD

⑦ Climates
 Winter: ↑↑ (مزيد من البرد)
 Summer: ↓↓ (مزيد من الحرارة)

Types of AD (Extrinsic & Intrinsic)

	Intrinsic (Non Allergic)	Extrinsic (Allergic)
• Incid.	Less Common (2%)	Common (80%)
• Age	Late (Adulthood)	Early (childhood)
• FH	Less +ve	Strong (+ve)
• ASS. other Atopic Manif. (diathesis)	Rare	Common ↑
• Mechanism:	Non Immune Mediated.	Immune Mediated. (Type I)
• Triggers	No — Food Aeroallergens Irritants	Food, Aeroallergens & Irritants.
• IGE • Cytokines: - IL4 & 13 - IL5 & IFNγ	NL ↓↓ No difference ↑↑	↑↑ ↑↑ No difference.

• Intrinsic:

(No)

→ Ichthyosis

→ Hyperlinearity

→ FLG →

NL barrier

• Epidemiology: Incid. AD affect (15-20%) of children & (1-2%) of adults.
 • Age: (85%) of cases appear in 1st year & (95%) appear ≤ 5 Ys.
 • Sex: M:F = 1:1.4

• CIP → 4 Phases

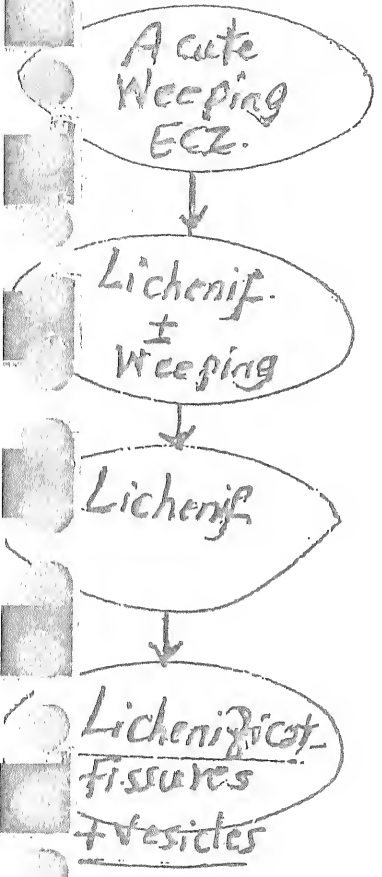
1. Infantile (2ms - 2 Ys)

2. Childhood (3 Y - 11 Ys)

3. Adolescent / Young adult (12 - 20)

4. Adulthood (> 20 Ys)

CIP ^{Age distribute} ^{Morphology} ^{clearing}
(4 phases) →



Atopic dermatitis may present at any age, but 85% of patients experience their first outbreak by their first birthday, and 95% by their fifth. Four clinical phases are recognized:

1. Infantile (2 months-2 years)
 - Distribution: Cheeks (Fig. 8-1A), face and scalp, extensor surfaces of extremities (perhaps from crawling), and trunk
 - Morphology: Erythema, papules, vesicles/oozing, and crusting
 - Clearing: Dermatitis clears in half of the patients by 3 years of age
2. Childhood (3-11 years)
 - Distribution: wrists, ankles/back of the thighs, buttocks/and antecubital and popliteal fossae (Fig. 8-1B)
 - Morphology: Chronic, lichenified scaly patches and plaques that may have crusting & oozing (see Fig. 8-1B)
 - Clearing: Two thirds of patients clear by age 6
3. Adolescent/young adult (12-20 years)
 - Distribution: Face, neck, arms, back, and flexures (Fig. 8-1C)
 - Morphology: Thick, dry, lichenified plaques without weeping, crusting, or oozing XX
 - Clearing: 90% or patients clear by age 18
4. Adult (>20 years)
 - Distribution: Most commonly involves the hands, sometimes the face and neck, and rarely diffuse areas
 - Morphology: Lichenified plaques, fissures on the hands, occasional vesicular outbreaks, one subset of "sensitive skin" patients

50% → ch
60%
90%

Infantile stage:

- XX - Diaper Area usually spared
- Exacerbation may occur d.t.
 - Emotional stress
 - Teething
 - RTI

NB
Dermatitis
< 3ms
→ SD
But > 3ms
→ AD

→ Hipp's Atopic Erythroderma, progression of condition to affect the whole body.

Childhood phase:-

- Fossae < Antecubital Antipopliteal
- This ECZ is a mixture of:
 - Prunigo of Hebra → Extensor of
 - " " Besnier → Flexor of

Adulthood Phase:

- Persistence > 30 Ys → usually in psychotic pt.
- dissemination may occur → disseminated ND.

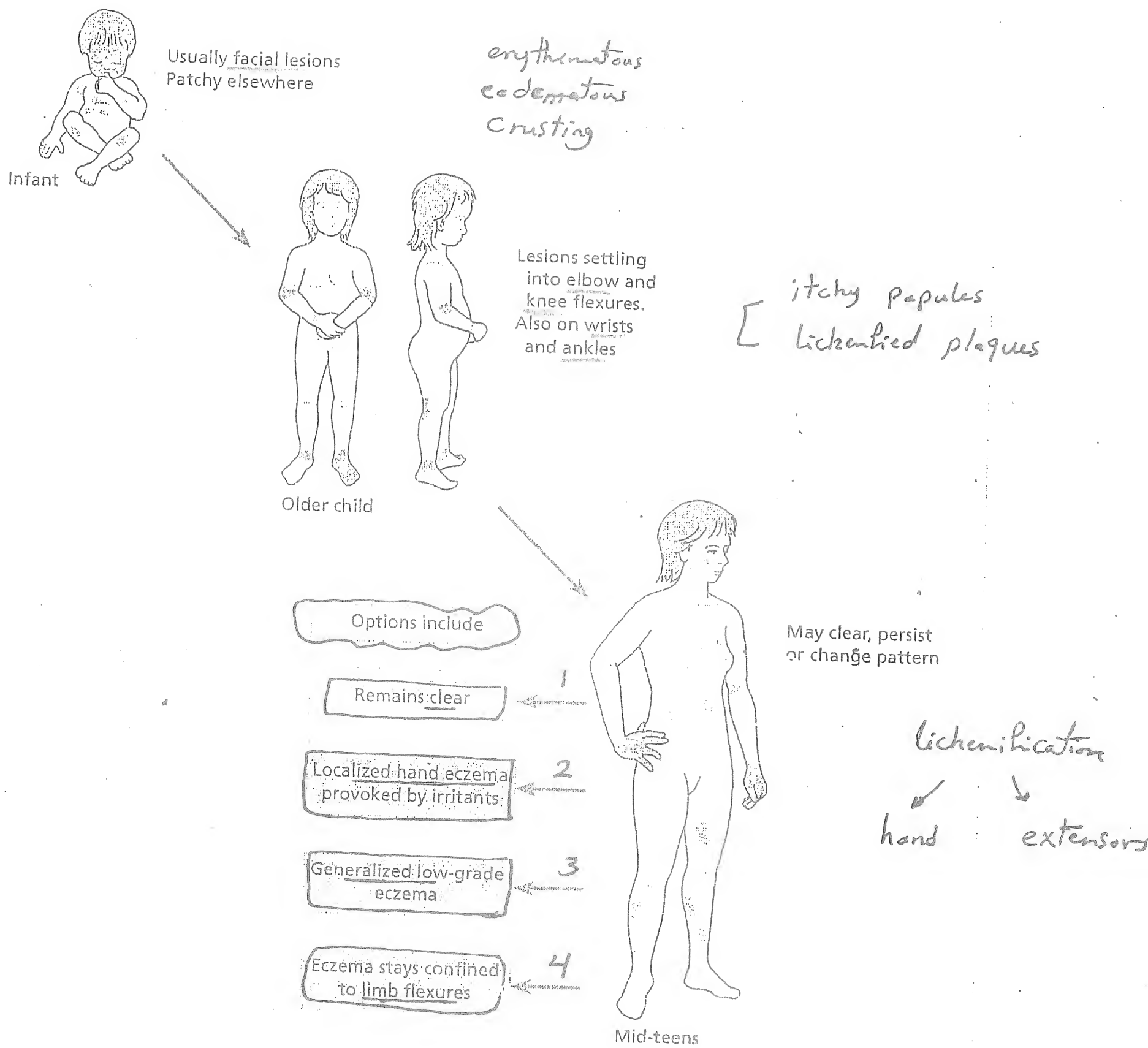


Fig. 7.12 The pattern of atopic eczema varies with age. It may clear at any stage.

Atopic erythroderma :-
 1st few days of life
 adults
 (Un Common)

Diagnostic Criteria For AD.

4

2 tables

- ① Hanifin & Rajka (1980) → the most accepted & widely used.
- ② American Academy of Dermatology Criteria (2003)
- ③ Williams Criteria / UK Party ^{Working} (2006)

A Hanifin & Rajka

هانفين
راجكا

A Major Criteria (4)

العرض الأساسي

Cardinal
Symptoms

1. Pruritus
2. Typical Morphology & distribution < ^{infancy}: Facial & extensor involvement
3. chronicity or chr. relapsing course < ^{Adults}: Flexure
4. Personal or family history of Atopy. (+ve) FH

ASSOCIATED FEATURES OF ATOPIC DERMATITIS

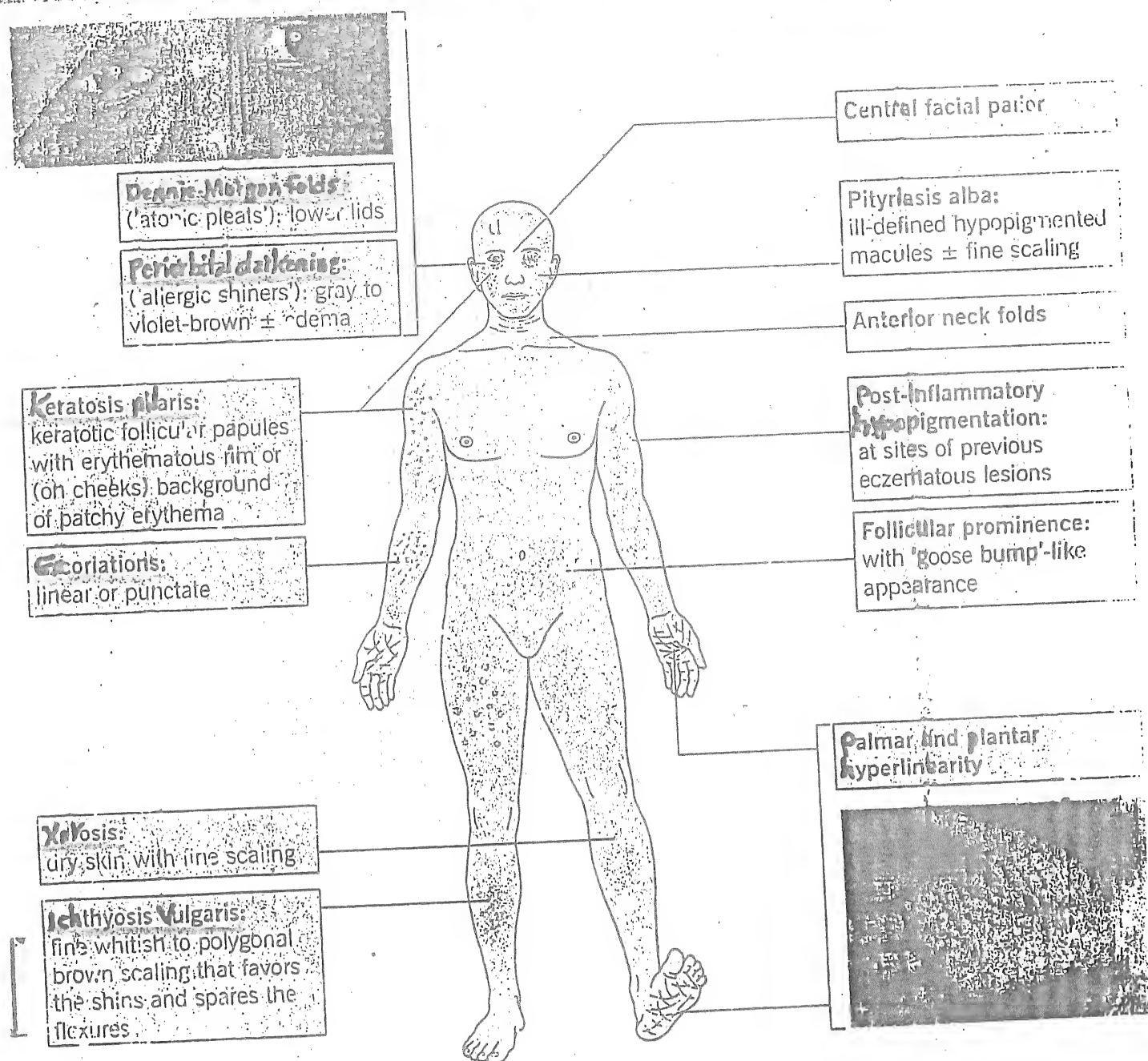


Fig. 10.10 Associated features of atopic dermatitis. inset of hand: Courtesy, Jean L. Bolognia, MD.

B Minor Criteria || "23"

- onset : Early < 2m.
- course : Influenced by < Environmental
Emotional Factors

Intolerance to Wool & Lipid Solvents.

IgE changes (\uparrow level & Reactivity)

Associated Features: "3"

for $D = 3$ Major + 3 Minor

Associated Features

Cut.
Eye
Stigmata.

1. Cutaneous:

- Xerosis
- Ichthyosis (↑ TEWL) due to xx FGL gene
- pit. Alba
- Keratosis pilaris (kp = 40%)

Reticulate pigmentation of Neck (dirty Neck)
periorbital milia

- White Dermographism
- Cholinergic urticaria ✓

↑ susceptibility to Inf < Staph. aureus.

2. Eye

- Facial Erythema / pallor
- Perifollicular Accentuation

HSV (Eczema Herpeticum)

- Hand Eczema
- Nipple II

- Cataract
- Kerato Conus
- Atopic conjunctivitis

3. Atopic Stigmata: → are permanent (دائم)

Eye

periorbital Hyperpig. (Allergic shiners)
Dennie-Morgan infra orbital folds (pleats)
Thinning of outer Eye brow (Hertogh's Sign)

Nose

Perinasal pallor (Head light Sign)
↑ Nasal creases

Hair

low set hair line. كت ابيض

Neck

Ant. folds

Palms

↑ creases. (Hyperlinearity)

6

B. American Academy of Dermatology (2003)

I. Essential features (must be present)

(must)

2/8

1. Pruritus

2. Eczema with:

Typical morphology, distribution and age specific pattern:

a. scalp in infants → checks ~

b. facial, neck, and extensor involvement in children

c. current or prior flexural lesions in any age group

xx [d] sparing of groin and axillary regions

3. Chronicity or chronic relapsing course.

II. Important features (seen in most cases, adding support to the diagnosis)

(most)

1- Personal or FH of Atopy.

2- Early onset < 2ms.

3. IgE reactivity.

4. Xerosis

III. Associated features (nonspecific clinical associations that help in the diagnosis of AD)

1. Atypical vascular responses (eg, facial pallor, white dermographism, delayed blanch response)

2. Keratosis pilaris/hyperlinear palms/ichthyosis

3. Ocular/periorbital changes

4. Other regional findings (eg, perioral changes/periauricular lesions)

5. Perifollicular accentuation/lichenification/prurigo lesions

→ all ass. features except (See Ass. Features)
x - Xerosis
✓ - Add vascular changes

IV. Exclusion criteria: presence of other conditions of Itchy skin e.g. CD & Scabies

- Patients must have an itchy skin condition (or parental report of scratching or rubbing in children)
- Patients also must have 3 or more of the following criteria:
 - History of flexural involvement, e.g. antecubital area, popliteal fossa, fronts of the ankles, or neck
 - Personal history of asthma or hay fever or a history of atopic disease in a first-degree relative in patients <4 y of age
 - History of generally dry skin in the last year
 - Visible flexural dermatitis or dermatitis involving the cheeks or forehead and outer limbs in children <4 y of age
 - Onset younger than age 2 years (not used if child is <4 y)

NBs:

1) Other Presentations of AD

- Nummular dermatitis
- Eyelid dermatitis
- Ear dermatitis; post-auricular fissures
- Cheilitis
- Nipple dermatitis
- Facial dermatitis
- Hand dermatitis
- Juvenile plantar dermatosis + (Sole)

Dermatitis of: -

- Face
- Eyelid
- Ear, postauric
- Lip
- Nipple
- Hand
- Sole
- Nummular

2) AD Associated diseases & Syndromes: -

A. Ass. diseases: -

- other atopic diathesises (30%)
- Eye → Cataract & Keratoconus
- Ear → OM
- Skin → Ichthyosis
- Vitiligo
- AA
- geographic tongue

B. Ass. Syndromes:

- Ataxia Telangiectasia (Ataxia + Telangiect. + Sinopulm. inf + Leuk. or lymph)
- Anhidrotic Ectodermal dysplasia (Anhid. + Alopecia + dental defect)
- AEP (Dermatitis + Diarrhoea + Alopecia)
- PK (↓ pigm. of skin + Hair + Deafness) = pigmentary dilution + Atopy
- Netherton synd. (Ichthyosis + AD + Trich. invaginata + FIT)
- omens synd. (FIT-EW ↑, ↑ IgE, ↓ IgA + Cut. + Eryth. + Syst. + L.N. + HSM + urine. diathesis)
- Hortnup dis. (vit. plagra + Neuro psychiatric + AA + ↓ urine. diathesis)
- Histocytosis X (niacin) - Wischott-Aldriche
- Job Synd. & Hyper-eosinophilic Synd.

3. In atopic dermatitis, which comes first—the itch or the rash?
No primary skin lesion has ever been established in atopic dermatitis. Hanifin and Rajka suspect that all the cutaneous changes of atopic dermatitis may be due to friction and itch-induced scratching. As Beltrani states, "Atopic dermatitis is an itch, which, when scratched, erupts." If there is no friction or scratching, there is no eruption.

Itch $\xrightarrow[\text{cause}]{\text{which}}$ Rash

What is the
1ry lesion
of
AD??

سؤال

4. SCORAD Index = Scoring AD :-

A. Extent criteria

B. Intensity :-

- Erythema
- Edema
- oozing
- Excoriation
- Lichenifications

كل واحد على حدة درجة
نقطة ال score
من 1-3

C. Subjective symptoms:

- pruritus
- insomnia

$$\text{Total score} = A/5 + 7B/2 + C$$

سؤال



Hyper-IgE syndrome (Job's syndrome) occurs in infants and children. It resembles atopic dermatitis but involves particularly the scalp, axillae and groins. The associated features include persistent secondary bacterial infection, fluctuant cold abscesses, contact urticaria, bronchitis and severe lung damage. The key laboratory finding is the great elevation of serum IgE (greater than 2000 i.u / mL) often with some eosinophilia. Neutrophil chemotaxis is impaired.

The hyper-eosinophilic syndrome is a rare disorder, occurring in middle aged males, and characterized by intense blood eosinophilia. There is affection of internal organs (heart, nervous system, Liver, lung and gut). The skin changes include an eruption resembling atopic dermatitis or urticaria, itchy maculopapular eruption or erythroderma. Treatment includes systemic steroids and cytotoxic drugs such as hydroxyurea.

- AD, AR or sporadic

- Mutated in \rightarrow STAT3 & Tyros. K2

النسبة الفوقانی

- 3 Lab \leftarrow $\begin{matrix} \text{IgE} \uparrow > 2000 \text{ i.u} \\ \uparrow \text{Neutrophils} \\ \text{Eosinophilia} \end{matrix}$

① AD with Bad Prognosis:-

- Young age
- ♀
- (+ve) FH
- other Manifests. of AD (diathesis)
- Persistent Xerosis in old age.

- Complications of AD:-

1. Impetiginisation & 2nd bact. inf.

2. Folliculitis

Q. إصابة → 3. Eczema Herpeticum (Kaposi varicelliform Eruption)

4. Ass. diseases & synds.

5. GR: in severe cases > 50% of BSA.

- Investigations

A - Skin prick test

[- Total IgE (RIST)

[- Specific IgE (RAST)

- Eosinophilia; blood, lesions

[- Th2 cytokine profile; IL-4, IL-5, IL-13

[- Reduced Th1 response; interferon- γ , DTH to contact allergens

INF- γ

NBs

1- Dennie Morgan folds:-

- Is d.t. Eyelid Edema ^{& itching} → Existence of 2nd or 3rd Palpebral crease.

- (+ve) in 75% of Atopics 5% of Normals → so of Little Value in Diagnosis

2. Some authors considered infraauricular folds & fissures are diagnostic because they are (+ve) in 22% of Atopics 33% of NLs.



Management of AD

(No Curative tt)

Q

10

1- General Measures :

عموم التدابير

- Bathing
- Clothes
- Exercise
- Environment / irritants & Contacts — exclusive breastfeeding 4-6m
- Diet (probiotics — prebiotics) — low allergen diet for mother during breastfeeding.
- Health Care Education

2. Topical tt :-

- Cs
- Cs + Antibiotics
- Calcineurin inhibitors
- Emollients
- others — Antipruritic
 1% di Na Chromoglycate

3. Systemic tt :

- Antihistamines ✓
- Antibiotics ✓
- Cs
- Tacrolimus
- Interferon
- Cyclosporine
- Rom & Cine
- Zafirlukast
- Anti PDE
- IVIG
- others — MTX
 Azathioprine
 Mycophenolate

4. phototherapy

5. others therapies

A. General Measures

1. Bathing

① الماء : يفضل لبانيو ١٠-١٥ دقيقة

استخدام ماء فاتر (tepid) [تجنب استخدام الماء البارد]

(جلد يكرس)
= hydration of
st. corneum

Should be:

② المصابون :

① Non irritant e.g. Dove, Keri, Nertingena

② acidic soap (grain Axilla) → staph

③ Antiseptics may be added to water as chlorhexidine or كلوركس Na Hypochlorite or

④ Soap used at Grion axillae: anogenital scalp.

1/2 cup for Full Tub or 1/4 cup for 1/2 Tub. (5mL → 4L)

إضافة حاجتين
ماء لبانيو

- Cetylote
- فنجات
emulsifying
oil

⑤ إضافة مرطبات طلاء لبانيو : (emollients)

- as mineral or vegetable oils

- Bleach bath زيت زيتون

⑥ اللبنة : (تكون اسفنج)

XX. Avoid Rough ones Vigorous Rubbing.

⑦ بعد الحمام : يفضل ترك الجسم يذوب تنشف أولفة بفوطه ناعمة قبل استخدام المرطب [لا بد من استعماله على طبقة ماء]

تجنب الجفاف بالفوطه أو استخدام (Hot air dryers)

استخدام مرطب الجلد [في خلال ٣ دقائق]

باستخدام [Soaking & greasing Technique]

فازلين Petrolatum oint

- fatty acids
- Linoleinic
- Prim Rose oil
- Ceramide

يفضل استخدام مرطبات مثل

[Oint base]

فوق طبقة رقيقة من الماء

حلمة تقطع
2 Pegam-
as
##

??

2. Clothes:

- Avoid ^{Wool} Textile fibres
- Use ^{Cottons} Linens (الكتان)
- Light & Not tight
- إزالة العلاقات مع الجلد
irritation ← [بلى عارضة]

→ mild - non alkaline cleansers

3. Exercise: ^{Regular &} in warm place

4. Environment: ~~Excess dry~~ // warm Environment. (Best) ^{Temp = 18°C} Humidity 50

5. - Avoid contact with irritants & sensitizing substances:-

- Antihistamines (Allergex cream)
- Neomycin
- Exacerbation of lesion on Topical Cs.

6. Diet: →

Learning point

Do not encourage cranky dieting for atopic eczema: it causes anxiety and seldom if ever does much good

7. Health care Education: Psychological Relaxation & avoid Emotional stress.

مع الأول
↓
بيجامه مبلولة
↓
بيجامه ناشف
فوق

under occlusion

Describe the "two-pajamas treatment."

One especially effective method for applying topical anti-inflammatory medications under occlusion is known as the "two-pajamas treatment." It involves taking two pairs of cotton pajamas and soaking one pair in tepid water. At bedtime, a mild- or moderate-strength corticosteroid or calcineurin inhibitor is applied to the involved skin immediately after bathing. The wet (wrung-out) pajamas are then donned, followed by the dry pair. These are worn through the night. In the morning, this treatment can be repeated, or the patient can bathe and immediately apply emollients and clothing. This type of therapy can be modified as the "two-socks," "two-gloves," "two-caps," "two-shirts," or "two-pants" treatment as the distribution of lesions dictates.

Topical Therapies

1. Cs

شروط استعمالها في حالات AD

Weekend Superpotent better > daily potent

A. Ointment :

- No ACD (less preservatives < Cream)
- provide Superior Vapour barrier (moisurizing effect)

B. Potency:

infants → low potent Cs (Hydrocort. 1-2.5%)

children →

- Body → moderate potency
- Face → Elidel or weak potent Cs
- thick LSC & lichenif →
 - daily → mild Cs
 - Weekly → super potent.

Monitor for growth parameters.

C use 2 pajamas Technique:-

نوع

oral Cephalexin

intranasal Mupirocin oint 1-3 ms

2 Antibiotics : may be added to Cs

in cases of Acute Flare & act as Antistaph & steroid sparing

Antibiotics if.

overt clinical inf.

during Exacerbation

Atopic lesions Highly Colonized by staph

cheek cream Fucidort (كروتين)

③ Topical Calcineurin Inhibitors (TCI): (Tacrolimus & Pimecrolimus)

used if there is:-

1. Cs Resistance

2. C.I of Cs

3. Cs S.E

→ Face & glion to avoid atrophy
→ large surface area to avoid systemic Abs
→ Cs sensitivity

S.E: irritation so use Cs first then Elidel

④ Emollients:

• Petrolatum = فازلين

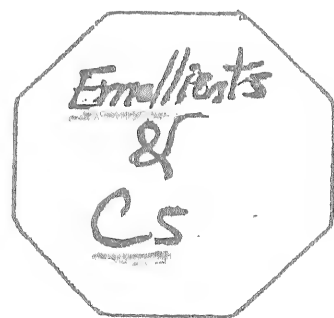
• 10% urea (كازامير)

• Crude Coal Tar in 1-5% white petrolatum.

⑤ Others:

- Classical Antiseptics
- 10% disodium Cromoglycate
- 10-30% Anhydrous Caffeine.
- Calamine lotⁿ.

Table 7.8 Winning ways with emollients.



Corner Stone
of
ADH

Make sure they are applied when the skin is moist
Prescribe plenty (at least 500 g/week for the whole skin of an adult and 250 g/week for the whole skin of a child) and ensure they are used at least 3-4 times a day
For maximal effect, combine the use of creams, ointments, bath oils and emollient soap substitutes

بالإشاحة
تسبب أضرار
من الحكة

??

Systemic therapy

لا فوائده
أفضل
من لقاحه

1- Anti Histamines :-

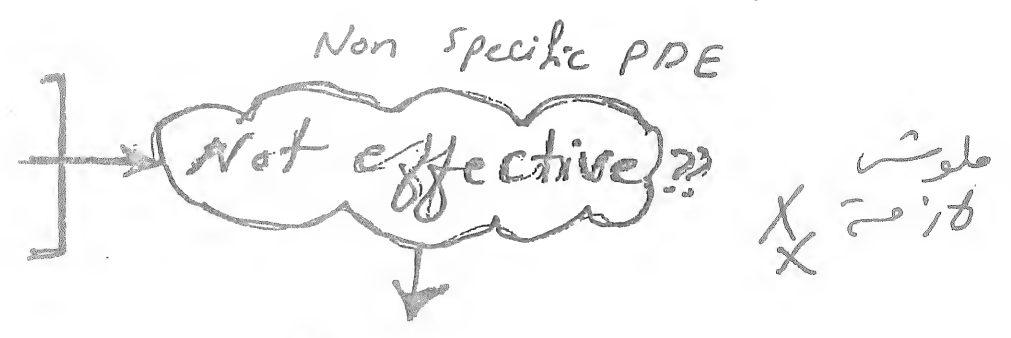
- Non sedating → No effect
- Sedating → effective by its sedating effect but not by its Antihistaminic effect (مفعول)
xxx

* Can be Combined :-

- Ketotifen → When suspected air borne Allergy
- di-Na Cromoglycate → When suspected food Allergy

2 PDE₄ inhibitors :

- X - papaverine
- X - Caffeine
- X - theophylline



كريزا برون
Crisaborole (Eucrisa)[®] Recent

- Topical (FDA 2016)
- Systemic (Not yet FDA)

Recently

- Imidazoleolidinone XX
- Ro 20-1724
- CP 80633

APX 80633
(Cotazila)[®]
PS

- ### 3- Others :-
- IVIG
 - MTX
 - Azathioprine.

Phototherapy

NB-UVB → 1st of choice for Severe AD.

Other tmts

- ← Hypnotic
- ← Herbal
- ← Thyroid

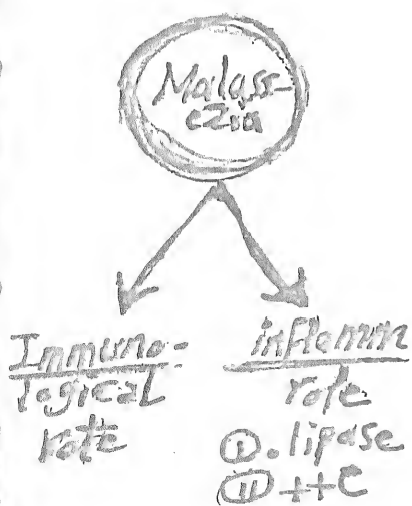
Endogenous ECZ.

Seborrhoeic dermatitis

def. Type of ECZ. (or papulosq. dis.) that affect the Seborrhoeic sites

Et & pathogenesis: unknown but ± d.t.

(hypersensitivity to Malassezia)



1-Seborrhoeic dermatitis is associated with normal levels of *Malassezia* but an abnormal immune response. *Malassezia* organisms are probably not the cause but are a cofactor linked to depressed Helper T cells, phytohemagglutinin and concanavalin stimulation, and antibody titers. The contribution of *Malassezia* species to seborrhoeic dermatitis may come from its lipase activity—releasing inflammatory free fatty acids—and from its ability to activate the alternative complement pathway. → ROS toxic effect

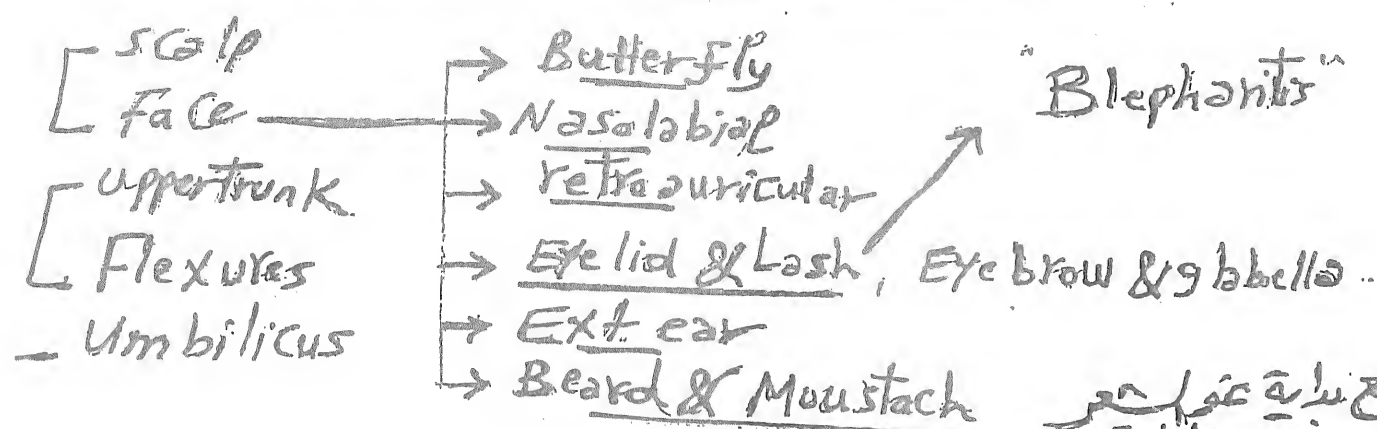
2- Persons prone to this dermatitis also may have a skin-barrier dysfunction (Invest Dermatol. Feb 2008)

↑ TG, ch
↓ Squamous, FFA

Epidemiology: Age: Any but commonest is at puberty (e peak at 40 Ys) & Neonates.

Sex & race: (No) predilection (but males slightly > ♀).

CLP: → Erythematous skin e overlying greasy scales that affect Seborrhoeic sites (Hairy areas) as:



Clinical varieties →

مع زيادة إفراز
دهون بالجلد
(Superficial form SD).

SD may be aggravated by:

- Trauma
- Stress
- Fatigue
- Seasonal changes & Humidity
- Drugs - Clotrimazole, Buspiron, Psoralens

griseofulvin
phenothiazine

(3P)

Sunlight → 1st ↑ then improved
↓ general Health

HIV
Parkinsonism
CHF

may → Erythrodermic SD

Table 17.3 Clinical patterns of seborrheic dermatitis.

18	Infant (Chapter 14)	cradle red
18	Scalp (cradle cap)	
	Trunk (including flexures and napkin area)	
	Leiner's disease	
	Non-familial → generalized SD self-limiting	Leiner's disc
	Familial CS dysfunction	
	Adult	
	Scalp	
	Dandruff (non-inflam.)	
	Inflammatory—may extend onto non-hairy areas (e.g. postauricular) (Corona Seborea)	Sebops
	Face (may include blepharitis and conjunctivitis)	
	Trunk	
38	Petaloid	
28	Pityriasisform, psoriasiform.	
	Flexural	
	Eczematous plaques	
	Follicular (DD-Dotier)	
	Generalized (may be erythroderma)	

Immune & dis order

- (de) effect of androgen from mother
- Seb dermatitis skin rash
 - diarrhea
 - FTR
 - Latent

"for overlap cases"

Some NBs:

① petaloid form (petal like):

as SLE

ago

① affect pre-sternal & interscapular areas

piel nei
well nei

② start as: Erythematous follicular, scaly, greasy

Papules → Coalesce → Figured Circinate Eruptions.

② Pityriasisform ch-By:

more

Generalized Ext. slow
lized inside resol.

Generalized & more extensive > pit. Rosea.

in particular affect Neck upto Hair lines

Resolve spont but slower > pit. Rosea.

③ Dandruff (pit. Capitis or sicca): 2 pit oleosa

Non inflammatory SD (1st degree of S.D)

chr. Cases of scalp dandruff & SD ± → Hair loss.

4. Severe SD may be complicated by Eczema Else Where:

[Pompholyx
Discoic EcZ.

5. Paranasal Erythema ± Flushing in Women ± → SD or Rosacea.

* Histopathology → Non Specific used to Exclude other conditions as PS

- Hyperkeratosis
- Acanthosis
- Spongiosis
- Accentuated Rete.
- ± Neut. infilt.

of SD

A

تعليمات المريض:

1. ## is for control Not cure.
عشان كده لازم توافيق على العلاج (ممكن مريض)
2. ممنوع استعمال:
[Alcohol based solutions xx
[Hair tonics. xx
3. غسيل الشعر بالماء وصابون بامترا.
مفيد بيه
✓ Remove lipid
✓ substrates of Malassezia.

B

Topical ##:

1. Topical antifungals → as in TVC ✓
shampoo (Zn pyrithione)
creams (Tar, Selenium Sulphide)
↓
Ketoconazole cr
2. Topical Cs:
سكروين لاستعمالها

ضعيف
ولادة قليل
Weak (Hydrocortisone 0.5%)
Short course

To avoid:

recurrence
↓
dependance & rebound.

3. other Topicals:

severe
→ 5% salysilic acid cr

- Calcineurine inhibitors
- [Imidazole 1% gel (2008) ←
- [Benzyl peroxide 5%
- [Propylene glycol.

C

Systemic therapy:

= syst Antifungal

- 1- Resistant Cases → Ketoconazole (1X1X14) اسبوعين
Itraconazole (1X1X21) ١٢ لايح
Lamisil (1X1X14) (1X1X14)
2. Generalized Severe: → Prednisolone 30 mg 1d. = syst cs
Retinoids c

D

Phototherapy: (UVB) for resistant cases.

Stasis Dermatitis

(Gravitational = Varicose ECZ.)

Def.: Stasis dermatitis is a common inflammatory skin disease that occurs on the lower extremities in patients with chronic venous insufficiency with venous hypertension.

قراءة سريعة

Pathophysiology: الخطوات التالية

↓↓ valve competency (Disturbed function of the 1-way valvular system in the deep venous plexus of the legs).

d.t

① Aging.

② DVT

③ Surgery

④ Traumatic injury

(stripping or saphenous V. harvesting for coronary by pass).

Chronic venous insufficiency (backflow of blood from the deep venous system to the superficial venous system).

Venous hypertension

Stasis dermatitis

There are 2 Theories Explaining the mechanism by which the Venous HTN can cause cut. inflamm. of Stasis Dermatitis:

① Stasis / Hypoxia theory: Stasis of Blood inside superficial venous system → Hypoxic damage of overlying skin.

← Not Accepted because leg veins in those patients were shown to have ↑ Flow rates & = not stasis

High O₂ Tension. (unknown Et).

بسبب هذه لنفرا حادثة لتيه
فالسفن برفه استقام هذا علاج

② Fibrin cuff theory (dermal microcirculation theory):

- Venous HTN → ↑ permeability of dermal capillaries → Fibrinogen leakage → Fibrin → Fibrin Cuff formation around dermal capillaries.

الأم

Fibrin Cuffs around Dermal V's

5

Barrier

بجمل ما بين

Leukocyte

TGF B₁
ICAM-1
VCAM-1

Serves as barrier to O₂ diffusion to skin

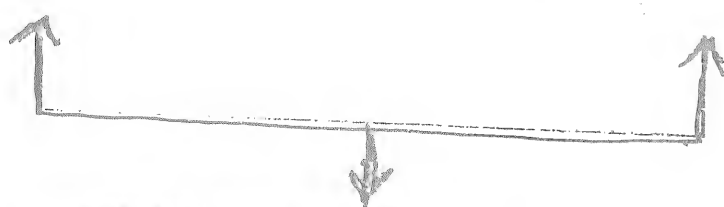
Traps activated Leukocytes

تمنع وصول
O₂
للجلد

Tissue Hypoxia & Cell damage.

TGF B₁ 1. Release of TGF B-1 (mediator for dermal fibrosis)

ICAM-1 VCAM-1 2. ↑ Expression of ICAM-1 & VCAM-1 (Potent chemottractants to keep Leukocytes in perivascular Environment).



proteolytic enz
Free radicals

Tissue damage

Cut. inflamm. & Fibrosis

HL → NB, 3 factors contribute in formation of fibrin cuffs:-

1. ↑ Permeability of Dermal Capillaries (d.t ↑ press)
2. ↓ Cut fibrinolytic activity
3. Leukocyte-Mediate cytokine products

- Dermal fibrosis is the hallmark of advanced stasis D.
- Fibrin cuffs not found in ulcers d.t causes other than Venous HTN.

- Henry et al (2001) → suggested that metalloproteinases may be important in lesional skin remodeling in persons with stasis D.

Epidemiology:

Incid: 6% in pts > 50 yrs & 20% in those > 70y

Age: usually > 50y

Sex: F+ > M (Effect of preg. on venous system)

علشان الحمل

Clp: The medial Ankle is most frequent & severely involved area (because it represent a water shed area with relatively poor Blood Flow compared with rest of the Leg); with advancement of the dis → encircling of ankle, Below Knee Extension (Stocking Erythroderma) & ± dorsal feet.

سایت
کانون
جانب

lesion →
 ① Reddish-Brown discoloration is the Earliest sign.
 ② There are Severe acute inflammatory, weeping Pathes & plaques that may be ass. with:
Honey-colored Crusting (dt bact.) or Monomorphous pustules (dt Cut. Candidiasis).
 - long standing lesions may show:

- 1- Hyperpig.
- 2- lichenification (Hemosiderin deposits)
- 3- Lipodermatosclerosis (Dermal fibrosis with inverted champagne bottle appearance).
- 4- PseudoKaposi Sarcoma = Angiodermatitis.
 - unique feature may be seen in stasis & ch'by Violaceous Nodules & plaque on dorsal feet that may undergo painful ulceration simulating "Kaposi Sarcoma"

x ③ Skin may show other venous insufficiency changes:-

- └ Edema
- └ Varicosities
- └ Atrophie blanche
- └ Hyperpig. (dt Hemosiderosis)

investigations:

- 1- Doppler studies → diagnosis of DVT & valve in competence
- 2- Histopath: [A] Acute & subacute lesions → spongiosis with superficial perivasc. infiltr.

- [B] Chronic:
- └ Acanthosis
 - └ Hyperkeratosis
 - └ deep dermal aggregates of siderophages
 - └ dermal fibrosis
 - └ intimal thickening of arterioles & venules

u)
Eczema
+
Hemosiderosis

Q^{NB}

diff. bet Pseudokaposi & Kaposi :

preexisting

pseudokaposi: Typical changes of stasis D.

+ capillary & fibroblast prolif.

Kaposi: Vascular slits + Atypical

endothelial cells + prolif. of vs independent of the preexisting one.

(افتكر نقل، يكتسب طرقة أولية) Treatment (2007)

Compression therapy

① by special stockings that deliver controlled gradient of pressure

② always Leg elevations

دائماً بيقع رافع، جله ف شاء
الجلوس أو الوقوف وحباً ما
يلبس إستراب (lowest leg)
Edema قبل ارتعال إستراب لازم.

Assessing pt. arterial circulation as if there is impaired

circulation → ↑ claudication & ± ischemic damage

Recent Ht

use of drugs that --
Neutrophil Mediated
cytokines Release as:-

① pentoxifylline

② PGE1.

Medical Ht

① A cute Weeping:-

ecz → Burket (K permanganate sol. + Mod. soap) → ligat.

Potent Cs oint

② Chr. stasis D →

calcineurin inhibitors

Why (No Atrophy & ↓ SE of Cs
No Tachyphylaxis)

① incompetent perforators

② Hemosiderosis

IPL (2008)

③ Ht of inf: Topical & systemic

Antibiotics

CD علاجاً Neomycin Bacitracin xx يارني

④ Systemic Cs: if there is systemic autoeczematization

⑤ long Term: Emollients ✓ under occlusions

لا حظ: شروط استعمال ال Cs هنا

① oint

② Mod. potency (super potent ← systemic absorption → Atrophy ulcer)

③ Not for long duration

Dyshidrotic Eczema

(Pompholyx, Vesiculobullous hand eczema)

Def: Dyshidrotic eczema is a recurrent or chronic relapsing form of vesicular palmoplantar dermatitis of unknown etiology. Dyshidrotic eczema also is termed pompholyx, which derives from cheiropompholyx, which means "hand and bubble" in Greek.

Etiology and pathophysiology: The etiology of dyshidrotic eczema is unresolved and is believed to be multifactorial. Dyshidrotic eczema is considered a reaction pattern caused by various endogenous conditions and exogenous factors: $\omega \pm$:

- * ① Genetic:
 - AD Familial pattern \pm present
 - Pompholyx gene in chromosome 18q22.1-3
- ② Atopy (50% of cases).
- ③ Dyshidrosis: Not a cause but associate it in (40% of cases & its ttt improves pompholyx. (aggravating factor)
- ④ Emotional stress.
- ⑤ CD (Nickel or Cobalt in diet).
- ⑥ septic focus e.g. strept or dermatophytide.
- * ⑦ Drugs: - Aspirin
 - OCPS
 - IVIG
- * ⑧ UVA phototherapy (2004)

may be reaction to fungus infection of feet = Trichophytid

NB: DD ① PP pustular ps. → the (1ry) lesion is pustules (not clear vesicles)

* ② chr. Vesiculobullous hand ECZ: usually (no) remission & the vesicles always present.

Epidemiology:-

- 3rd most common cause of Hand ECZ.
- usually affect middle Aged (≈ 38 Ys)
- M=F

CIP: Acute onset of Eruption of ^{غالباً حادة} Macropsopic, ^{نقش} Tapioca li deep seated Vesicles & bullae on NL background on Both Palms & soles & Sides of fingers.

Nail fold
± affected →
Nail dystrophy.

- Commonly there are 2 ss:-

"^{نقش}" → pruritus: may precede the Eruptn.
Hyperhidrosis

- Site:
 - Palms only → 80% (called cheirpompolyx).
 - Sole " → 10% (" pedo ").
 - Palm & Sole → 10%.

- Fate: outbreaks resolve spontaneous over several ws ($\geq 2-3$ ws) & Recurrence is common (5%) at intervals of 3-4 ws for ms - Ys.

NB - Uncommon unilat cases → suspect CD.
 - Pompholyx should be reserved for Typical cases in w attacks resolve & recur while chr recurrent vesiculatn without remission called chr. Vesicular dermatitis.

CD → unilat
chr vesiculobullous dermatitis
على طول موجود

Histopathology: - Spongiosis & epid. Lymphocytic infilt.
 - intraepidermal Vesiculatn

TH: [1] Remove possible Causes:

hyperhidrosis TH

[stress
Hyperhidrosis] [CD
Fungal inf]

المستحبات
Al. acetate

[2] acute cases → Drying antiseptic lotn Drainage of large bulla

[3] subacute & chr. cases → Cs oint, Emollients & Keratolytics

[4] Resistant cases → MTX & radiatn TH photochemo therapy.

Lissuring

Ref. All sources

Asteatotic Eczema
Eczema craquelé, Xerotic eczema, Chapping

Def. Eczema Characterized by pruritic, dry, cracked, and polygonally fissured skin with irregular scaling. It most commonly occurs on the shins of elderly patients, but it may occur on the hands and the trunk.

Etiology and pathophysiology:

Causes

↓ lipids & water of skin

*Multiple etiologic factors may coexist to cause asteatotic dermatitis, including the following: (All are associated with ↓ lipid content of skin):

↓ or water

↓ NMF Natural Moisturizing Factor

- Ageing: due to ↓↓ sebaceous and sweat glands activity and ↓↓ Keratin synthesis.
- ↓ humidity and cold: → increase the loss of water by convection.
- Wrong Behaviour: - Frequent or prolonged bathing in hot water and use of soaps, infrequent use of emollients and use of Degreasing agents (Solvents and Cleansers)
- Atopy
- Ichthyosis
- Radiation
- Drugs - Antiandrogen therapy⁵ and diuretic therapy
- malabsorption and Nutritional deficiencies of essential fatty acids, including linoleic acid and linolenic acid, Zinc deficiency³
- Thyroid disease - Myxedema and other thyroid diseases with diminished sweat and sebaceous gland activity⁴
- Neurologic disorders - Decreased sweating in denervated areas
- Malignancies - Malignant lymphoma,⁶ gastric adenocarcinoma,⁷ glucagonoma, angioimmunoblastic lymphadenopathy,⁸ breast cancer, large-cell lung carcinoma, and colorectal carcinoma²

دال scrub

Epidemiology: *Age: elderly >60 y.

*Sex M > F

c/p: Primary lesions: Slightly scaly, inflamed, curvilinearly cracked and/or fissured skin most commonly involves the pretibial areas, but it may also occur on the thighs, on the hands, and on the trunk (Fitzpatrick likened asteatotic eczema to a dried-up riverbed).

الأرض المصقوفة

يهرج من قبل

شبه

Secondary lesions: Excoriated, erythematous, edematous patches may result from rubbing or scratching.

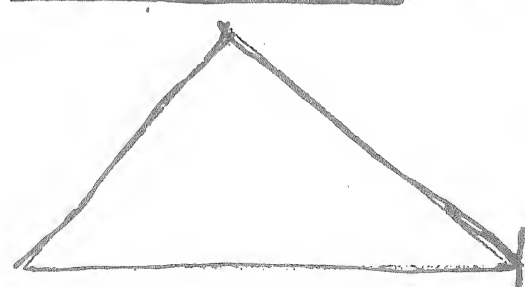
Clinical types: 1-localized: usually pretibial

2-Generalized (Ichthyosis): ?? Mg

III

1 تعليمات للمريض : الحمام بفاعك لازم

- 1- لفترة قصيرة (10 دقائق)
- 2- بفاع فاستر
- 3- بلاش صابون
- 4- بلاش صوف
- 5- استخدم Humidifier



3 Topical Cs

Emollients:

استعماله بعد الحمام مباشرة (AD)
استعماله يوميا 3 مرات على الأقل

Soaking and Greasing: (Soak & smear) Technique

Soaking: of affected part in Tepid Water for 10 min

Greasing (Smear): Immediate use of mild Cs oil

انقع في المياه 10 دقائق
ادهن على طول

Mild Topical Cs (III-IV)

يمكن استعمالها لبرهة من الزمن

A Soaking & Greasing

B underocclusion for 1-2 ds

Nummular Eczema

(Discoid Ecz.)

14

Def. Type of Endogenous Ecz. ch by:

Well defined, Coin-shaped, scaly, plaques
usually on arms & legs. w are very itchy &
very persistent

• AET & pathophysiology :-

Atopy 1. AD (recently considered as Adult onset AD)

2. Infect: (staph usually) colonize or infect it).

3. Emotional stress.

4. local & physical Trauma: lyc Intc

- Insect bite

- Abrasions

- CD

5. Xerosis → disturbed barrier → ↑ Penetration of
Allergens → CD.

6. Alcohol Abuse.

drugs = 7. Generalized form Recently ass e:

IFN α for hepatitis C
Ribavirin.
Anti TNF α .

8. Autoeczematization
(Discoid Ecz. is a type)

C/P: ① Age: 2 peaks

60-70y (++)
20-30y (*)

Clp : Well demarcated Coin shaped plaques of closely set, thin walled Vesicles on an Erythematous base or are dull red, oozy → this may be followed by Central clearing & Peripheral Extension → ring shaped or annular lesions may → dry scaly patches.

critically:
[Very itchy & Very persistent]

after any period bet lads - ms; (dry) lesions occur often in a mirror image configuration on opposite side of Body.

a very chic feature of ECZ. that: the patches w have apparently become dormant may become active again specially if it is discontinued prematurely.

3 patterns are recognized:-

1. Discoid ECZ. of hands & forearm
2. " " " Limbs & Trunk
3. DRY Discoid ECZ.

- Type 1 → dorsal Hands & Finger sides
- Type 2 → Seen in elderly & pts. w Xerosis.
- Type 3 → DRY discoid plaques on legs & arms, mild itchy (but not severe) & resistant to it

Course Waxes & Wanes For Years with recurrence in cold or dry climates

Is there a cure for nummular dermatitis?

No. However, the disease can be controlled. Many of the same principles apply here that apply to the treatment of atopic dermatitis. Limiting baths and soap exposure, avoiding irritants, frequent use of emollients, topical corticosteroids, avoiding dry environments, and antihistamines all have a role in treatment. Topical corticosteroids are the mainstay of therapy. With the high rate of staphylococcal colonization, many dermatologists routinely prescribe a 2-week course of oral antibiotics.

staph carrier and

such as dicloxacillin or cephalexin. Systemic steroids should be used only for severe cases and limited to a tapered course over 2-3 weeks. Severe chronic cases may also benefit from PUVA.

Does nummular dermatitis resolve spontaneously?

Yes, but not often. In a prospective study of patients followed for 2 years, 22% were disease-free. Another 25% were free of lesions for weeks to months, but 53% were free of lesions only with continued local therapy. If there is no clearing within 1 year, the disease tends to persist for many years.

Waxes & wanes
and is it

III

Juvenile planter Dermatitis

(DNNZ)
Androm
Reak (18)

def Eczema affecting the feet of children.

Aet: Repeated friction & Macerata of feet by: "الكوتش"

1. occlusive shoes specially Athletic shoes
2. Abrasive effect of pool surfaces or diving board
3. Thin Non-absorbant Synthetic socks.

4
Some consider
if manifests
of AD.

تعتبر ان سببها هو تغير الى جلد آخر بسببه في صناعة الأذية، وشرائط الأطفال
حيث تم استبدال المواد الطبيعية (الصوف، القطن، الجلب) بمواد صناعية (نايلون، بلاستيك)
والتي تعتبر أقل مسامية من المواد الصناعية، بالإضافة الى تغطية الأذية بمواد repellent
على أن تحسن من durability صناعة سطح كراء.

all these: ↑ Humidity → Feet macerata → sweat retention
→ aggravate the condition

ليبدو من كل لقطة لانه لو كان الى جلد طبيعي يكون بديل عن الجلد الطبيعي
(So role of friction Considered) ✓

CIP

Age: 3 Ys - Puberty (14)

2 Start as bilat, symmetrical red glazed
smooth patches at medial or dorsal
Aspect of Big Toe → then spread to
involve weight bearing areas of feet
as forefoot. (Heel less affected).

Note: webs & arches are spared.

DD: [AD [psoriasis
[CD [fungal inf.

Treatment: 1- Reduce friction: جزيمة مدر - صناعة القدم - شرائط
(porous foot wear).

ممكن تجنب
لواصها عن البلوغ

2. Emollient.

3. Have rest day: schedule quite time &
little or No walking
to allow for healing of fissure

4. Topical Cs

Hand eczema

تحریری

Hand eczema is such a common and distressing condition, and poses such difficult problems for the dermatologist, that it deserves separate consideration. Up to 30% of occupational medical practice relates to hand eczema, with important issues regarding medical litigation, worker's compensation and disability. One-quarter of the patients referred to a specialized contact dermatitis clinic suffered from hand dermatitis.

Classification: 1-Etiologic classification (Roz. K.) 2-Morphologic classification 3. Classification Acc. to the Age.

Most common exogenous cause →

Exogenous	Endogenous
<p>1-ACD:</p> <ul style="list-style-type: none">• Delayed hypersensitivity (type IV) (e.g. chromium, rubber)• Immediate hypersensitivity (type I) (e.g. seafood) <p>2-ICD:</p> <ul style="list-style-type: none">• Chemical (e.g. soap, detergents, solvents)• Physical (e.g. friction, minor trauma, cold dry air) <p>3-Ingested allergens (e.g. drugs, possibly nickel, chromium)</p> <p>4-Infection (e.g. following bacterial infection of hand wounds)</p> <p>5-Secondary dissemination (e.g. dermatophytide reaction to tinea pedis)</p>	<p>1- Atopic</p> <p>2- Dyshidrotic (Pompholyx)</p> <p>3. Psychosomatic (↑↑ Eczema > Initiation) → aggravate ecz</p> <p>4- Idiopathic (Discoid & Hyperkeratotic palmar ECZ).</p>

2-Morphologic classification

- 1-vesiculobullous hand eczema(Pompholyx) / & patchy vesiculosquamous
- 2-hyperkeratotic hand eczema / 7
- 3-dry palmer eczema = House wives ECZ = wear & tear dermatitis
- 4-finger tip eczema
- 5-ring eczema
- 6. Localized thumb ECZ
- 7-discoid eczema
- 8-chronic acral dermatitis
- 9-apron eczema
- 10-gut eczema
- 11-other patterns (eg.patchy vesiculosquamous)

not itchy
! — no vesicles
→ Recurrent local palmar peeling.

1- chr. Vesiculobullous Hand ECZ: see Pompholyx.

② Hyperkeratotic Hand ECZ (Tyrphic ECZ)

- Unknown Etiology
- M > F usually middle aged & over
- Clp → highly irritable scaly fissured, Hyperkeratotic patches on palms & palmar aspects of fingers.

حرف جراً
مركباً بالولوى
مكبراً من
مفرد

- DD: [palmar ps] → 'ch' Bx:

- silvery scales
- Sharply demarcated scalloped edges to the Erythema along borders of < Hands & Fingers.
- Absence of pruritus xx
- involvement of knuckles.
- Nail pitting (cut nail fold affects)
- other psoriatic sites.

- tt: (Extremely refractory to tt):

→ CS + Keratolytics

[PUVA
Etretinate

- Grenz Zone tt

③ Dry palmar ECZ = Housewives ECZ =

Wear & Tear dermatitis:

→ usually affect Housewives & cleaners d.t
Contact with water, detergents, soaps, solvents
& even food cuttings. (48/53)

- The skin looks Dry, criss crossed & superficial
Cracks ass. with damaged Horny layer &
unable to respond its Normal pliability to
hand & finger movement.

- There may be ass. with dorsal Hand chapping

④ Finger-Tip ECZ: - affect palmar aspect of finger tips usually localized but may extend to the palms.

clp - Finger tips are → dry, cracked, painful & fissured.

+ There are 2 Types Finger Tip ECZ

Type I (Common)
affect most of all fingers of
dominant hand specially
Thumb & forefingers

↓ its d.t
ICD (Cumulative)

Type II (less Common)
affects thumb, forefinger &
ring finger of dominant
hand → usually occupational

↓ ± d.t
ICD
ACD

⑤ Ring ECZ

→ accumulation
of solvent's
& soaps
under Rings

Cause:

• سبب تحت لدية أو الحاتم
نتيجة تراكم الصابون والمواد المنظفة وليس
حاسبة لاداة لدية أو الحاتم

Site: wedding ring finger & may extend to
middle finger.

⑥ Localized Thumb ECZ

- child who
put fingers in mouth.

في الإصبع (صغار الذين يقعون الأصابع في
فمهم أو يقرعون)

- may be assoc. → Nail dystrophy.

⑦ Discoid ECZ → see before

⑧ Chr. Acral dermatitis:-

chr.

- distinctive synd. chr by hyperkeratotic papulo-
Vesicular ECZ of Hands & feet intensely
pruritic & is assoc. with marked ↑ IgE in
middle Aged without personal or FH of Atopy
⑧ → Cs (systemic); Topical not effective
XX

ECZ.
+ ↑ IgE
↓
Cs systemic

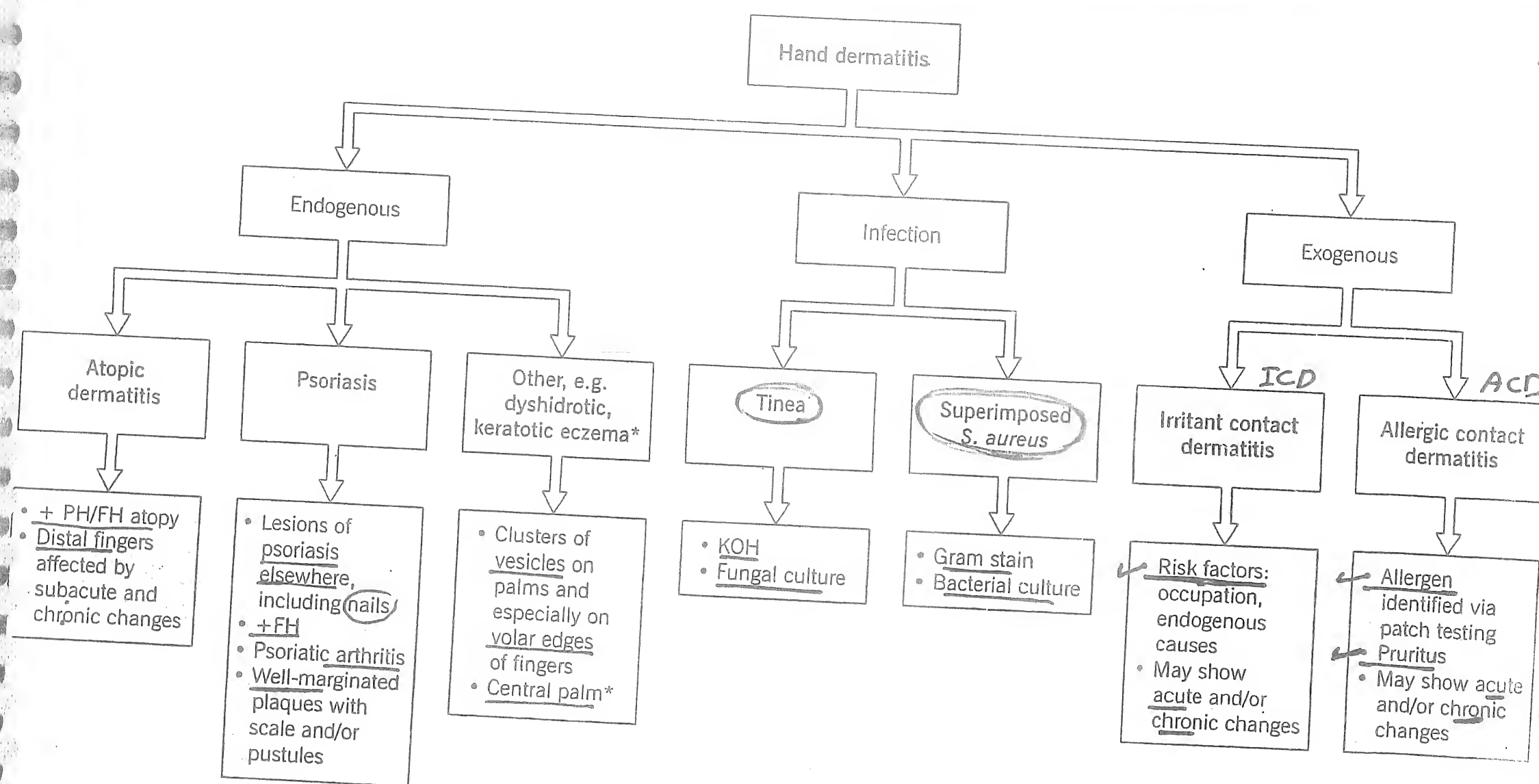


⑨ Apron ECZ : ECZ. Caused by ICD affects palmar aspects of adjacent fingers & forming in a circular pattern over MCP in a pattern - Resembling Apron.

⑩ Gut ECZ (Slaughter house ECZ):

- This type of ECZ affect workers who eviscerate & clean pig carcasses
- Clp: Vesicular ECZ. starts at fingerwebs & spreads to sides of fingers.
- Usually self limiting & clears in $\approx 1-2$ wks but may recur.
- Et: unknown but \pm d.t. (fat or blood ICD)

⑪ Patchy Vesiculosquamous ECZ : Bilat, Asymm. mixture of irregular, patchy, vesiculosquamous lesions



* some clinicians view keratotic eczema as a form of psoriasis

Lichenification

Introduction

Lichenification is a pattern of skin response to repeated scratching or rubbing, characterized histologically by acanthosis, hyperkeratosis, and elongated rete ridges, and clinically by a thickened skin, with accentuation of the surface markings so that the affected skin surface resembles tree bark. It may be primary (lichen simplex), without an itchy skin disease and caused by emotional tension, or secondary to an itchy skin disease as venous eczema, atopic dermatitis, chronic contact dermatitis, or chronic infection with *T. rubrum* of thighs or feet. & hyperpigment

Lichen simplex chronicus (LSC)

(localized Neurodermatitis)

Def. Reactive pattern of skin that arises (2ry) to repeated scratching or rubbing [so it is not a 1ry process] & ch- by cut. lichenification Δ of:

Δ { Thickening
Hyperpigment.
Accentuated skin
Markings.

Etiopathogenesis unknown but ±:

↓
• Emotional stress → Sensation of Burning or pruritus → Rubbing
→ Lichenification → More Rubbing → More lichenific.
(Viscious Circle of Itch/Scratch Cycle)

Epidemiology: Age: any but usually (30-50y)
Sex: ♀ > ♂

CIP

(1) itching: severe & occurs in paroxysms of great intensity → till soving.
there is (refractory period) of some hours before until itching recur.

(2) Skin lesion: - at first: Erythematous, Edematous Excoriated plaques classical picture of lichenification
- Lichenified papules ± seen.

• Site: Most Common sites are:

Nose Neck → [occiput
Nuchal area (Lichen Nuchae)] ⊙
— Perineum & Scrotum ⊙
[Wrist & ankle
Extensor forearms]

• NB: ① Giant lichenification of Pautrier

LSC in areas of loose S.C.T as Genito-Cervical area → Solid Tin like plaque & Warty Cribriform surface.

② Notalgia Parasthetica LSC at inf. tip of Scapula.

• Pathology → see lichenification

DD ① lichen amyloidosis ② L.p ③ ps.

treatment:-

1. Stop itching (Break the itch / scratch cycle):-

[Anxiolytic
وېلن کونکې دوا

2. Cs: Topical & ILs

3. Emollients

(2009)

(JAAD 2001)

4. Antihistamines, Botox, Topical Aspirin / dichloromethane

5. TENS (Transcut Electric N. Stim).

Other Types of Eczema

- Frictional lichenoid Dermatitis
- Eye-lid EcZ.
- Breast EcZ.

• Sandbox or Frictional Lichenoid Dermatitis.

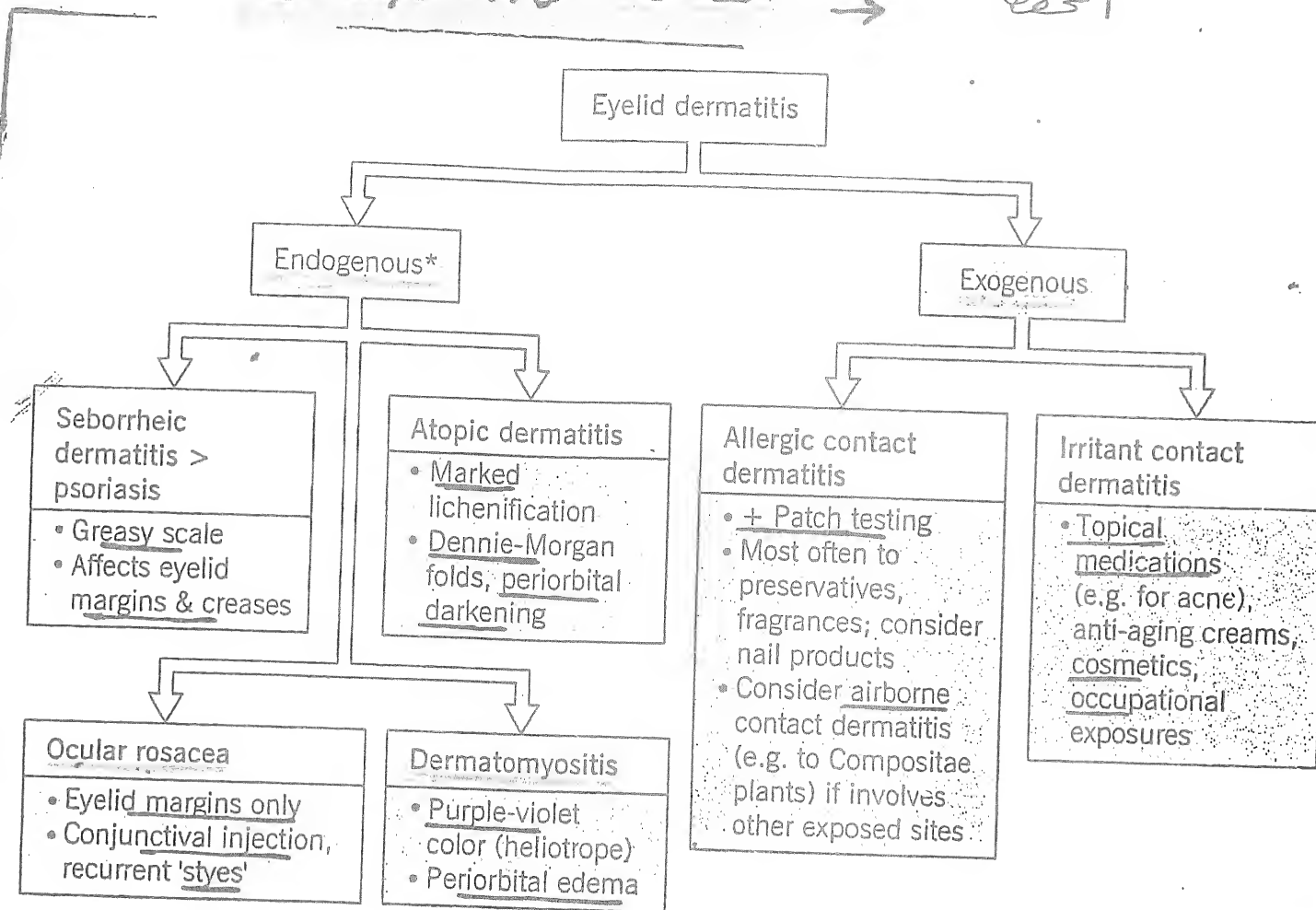
- Pin-head sized, white papules at friction sites

- Etiopath. ?? friction or sun.

- DD: lichen-nitidus.

(NB) In Adults: Dermatitis papulosa
Juvenilis

2. Eye-lid EcZ. →



*Diagnostic clues include a history of the condition and characteristic lesions elsewhere

Fig. 13.6 Classification of eyelid dermatitis. More than one etiology may be present, e.g. atopic dermatitis plus irritant contact dermatitis.

3. Breast Eczema (Nipple EcZ.)

- ± Affect Nipple, Areola & Surrounding skin. (specially of Nursing Mother).

- Etiology: CD, ND, AD, SD

- !!!: As EcZ. → if No response → Biopsy to Exclude "Mammary Pagets" ??

- Unilat.

- Not responding to CS

Acroangiokeratosis

Acroangiokeratosis

(Synonyms: pseudo-Kaposi's sarcoma, acroangiokeratosis of Mali-Kuiper, gravitational purpura, stasis purpura)

Definition: was first coined by Mali in 1965. [1] It is a proliferation of pre-existing vasculature seen in venous hypertension, arteriovenous malformation, or acquired iatrogenic arteriovenous (AV) fistula.

Etiopath. chr. Venous insuff → Venous HTN → Tissue Hypoxia → Neovascularization / Fibroblast prolif
C/P: Confluent, violaceous or brown-black papules cover large areas of the distal parts of the legs. Ulceration and bleeding are sometimes noted. Bilateral lesions are usually associated with chronic venous insufficiency, whereas unilateral lesions suggest an underlying vascular malformation.

Types:

- 1] Mali Type → ass. with Stasis Dermatitis
- 2] Stewart-Bluefarb Type → ass. with Cong AV malformation
 e.g. Klippel Trenauay synd
- 3] Dermite Ocre of Favre: ass. with pregnancy.
- 4] other Types:
 - ① AV Fistula of shunt in CRF / stump Dermatitis in Amputees
 - ② HcV ass. (E Med. 2010).

	Acroangiokeratosis of Mali	Kaposi's sarcoma
HP	Small dilated vessels lined by <u>plump</u> endothelial cells with hyperplasia of pre-existing vasculature	Slit-like spaces and <u>spindle cell proliferation</u> . Vascular proliferation is independent of the existing vessels
PAS	<u>+ve</u>	<u>-ve</u>
Factor VIII Ag in Endoth	<u>+ve</u>	<u>-ve</u>
CD34	Positivity seen on endothelial cells of hyperplastic vessels	Positivity seen on both endothelial cells and the characteristic spindle-shaped, perivascular cells
RBCs extrav Hemorrhage	Present	Present
Fibs		

Treatment

- 1- Correction of the underlying chronic venous insufficiency and vascular malformations
- 2- Systemic therapy: Various medical modalities of therapy have been tried with favorable results but options are limited. Oral erythromycin 500 mg four times a day
- 3- Dapsone
Topical Cs
- 4- Vascular lasers.

Dyshidrotic Eczema (Pompholyx, Vesiculobullous hand eczema)

Def: Dyshidrotic eczema is a recurrent or chronic relapsing form of vesicular palmoplantar dermatitis of unknown etiology. Dyshidrotic eczema also is termed pompholyx, which derives from cheiropompholyx, which means "hand and bubble" in Greek.

Etiology and pathophysiology: The etiology of dyshidrotic eczema is unresolved and is believed to be multifactorial. Dyshidrotic eczema is considered a reaction pattern caused by various endogenous conditions and exogenous factors: $\infty \pm$.

- * 1- Genetic:
 - AD Familial pattern ± present
 - Pompholyx gene in Chromosome 18q12.1-3
- 2. Atopy (50% of cases).
- 3. Dyshidrosis: Not a cause but associate it in 40% of cases & its improves Pompholyx.
- 4. Emotional Stress.
- 5. CD (Nickel or Cobalt in diet).
- 6. Septic focus e.g. Strept or dermatophytide.
- * 7. Drugs: Aspirin, IVIG & PUVA.

- CIP :: Acute onset of eruption of Bilat & symmetrical Deep seated vesicles & Bullae at Palms (Cheiropompholyx) Soles (Pedopompholyx) or Both. Nail fold may be affected
→ Nail dystrophy. ass. e severe itching & Burning.
- Exacerbation & Remission is Common (Chronic Vesiculo-Bullous Eczema)
- Unilat. Cases may be d.t. (C.D.) موجودہ شکل طور
- 3rd Most Common Type of Hand ECZ. usually affect middle Aged.

- ## 1- Treat the underlying cause.
- 2. Vesiculo-lesion → drying Antiseptic lot.
- 3. Chronic → CS, Emollient & Keratolytic
- 4. Resistant cases → systemic CS
 - MTX
 - Radiat

Nummular Eczema

(Discoid Ecz.)

(14)

Def: Type of Endogenous Ecz ch BY:

Well defined, Coin-shaped, scaly, plaques

usually on arms & legs w are very itchy &
[Very persistent

• AET & pathophysiology :-

"Atopy" ①- AD (recently considered as Adult onset AD)

②- Infectn (staph usually colonize or infect it).

③- Emotional stress.

④ Xerosis

C/p : Discoid plaques ch BY:

- May undergo central clearing → Annular lesion.

- Very itchy, Very chronic

- usually at: Forearms, legs, Hands, Lumb & Trunk

- Staph has a Marked Role → AB أحياناً

- More in Elderly (60-70ys)

Is there a cure for nummular dermatitis?

No. However, the disease can be controlled. Many of the same principles apply here that apply to the treatment of atopic dermatitis (Limiting baths and soap exposure, avoiding irritants, frequent use of emollients, topical corticosteroids, avoiding dry environments, and antihistamines all have a role in treatment. Topical corticosteroids are the mainstay of therapy. With the high rate of staphylococcal colonization, many dermatologists routinely prescribe a 2-week course of oral antibiotics

such as dicloxacillin or cephalixin. Systemic steroids should be used only for severe cases and limited to a tapered course over 2-3 weeks. Severe chronic cases may also benefit from PUVA.

Does nummular dermatitis resolve spontaneously?

Yes, but not often. In a prospective study of patients followed for 2 years, 22% were disease free. Another 25% were free of lesions for weeks to months, but 53% were free of lesions only with continued local therapy. If there is no clearing within 1 year, the disease tends to persist for many years.

قواعد عامة

← staph Carrier fl8
ABS

Topical Cs

Emollients

Antihistamine

Syst Cs

PUVA

##

Late

(EMat 2009)

Pityriasis Alba

(10)

def: Non specific dermatitis of unknown Aetiology that causes Erythematous scaly patches → These resolve & leave areas of Hypopigment that slowly repigment to NL.

Aetiology: unknown but ± d.t:

- assé
- 1. Atopy
 - 2. strept. inf.
 - 3. Sun Exposure (ظروف)
 - 4. ↓ Zinc
 - 5. ↓ Fe
 - 6. Parasitic infestations.

7. Malassezia. produces a substance² called pityriacin → sun filtrate (prevent Natural sun Tanning).
Topical Antifungal
air 1/5

CIP: ① usually affects dark skin children at summer
② has (2) stages: ١٨

Early: ill defined pink erythematous, scaly lesions →
Late: Hypopigmented (ill defined & scaly)

③ usually at sun exposed areas

④ Clinical Varieties:

① Classical Type: at sun exposed areas

② Generalized = Bilat & symm.

③ Pigmenting = Central bluish pigmentated surrounded by ill defined slightly scaly Halo at the face.

Treatment: ① Treating the cause e.g. Sunscreen, Parasites, Vit. deficiency

عوب جرد
وبو جرد

② Early stage → Hydrocortisone 1%
Late → Emollient

③ Some Authors: Ezalline (Similar to Vitiligo ttt) →

SE 74
hyperpig

Hand eczema

Hand eczema is such a common and distressing condition, and poses such difficult problems for the dermatologist, that it deserves separate consideration. Up to 30% of occupational medical practice relates to hand eczema, with important issues regarding medical litigation, worker's compensation and disability. One-quarter of the patients referred to a specialized contact dermatitis clinic suffered from hand dermatitis.

Classification: 1-Etiologic classification (Rat. K.) 2-Morphologic classification 3- Classification Acc. to the Age 1-Etiologic classification

Exogenous	Endogenous
1-ACD: -Delayed hypersensitivity (type IV) (e.g. chromium, rubber) -Immediate hypersensitivity (type I) (e.g. seafood) 2- ICD: -Chemical (e.g. soap, detergents, solvents) -Physical (e.g. friction, minor trauma, cold dry air) 3- Ingested allergens (e.g. drugs, possibly nickel, chromium) 4- Infection (e.g. following bacterial infection of hand wounds) 5- Secondary dissemination (e.g. dermatophytide reaction to tinea pedis)	1- Atopic 2- Dyshidrotic (Pompholyx) 3- Psychosomatic (↑↑ Stress > Initiation) 4- Idiopathic (Discoid & Hyperkeratotic palmar ECZ.)

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Id

2-Morphologic classification

- 1-vesiculobullous hand eczema (Pompholyx) / Patchy vesiculosis.
- 2-hyperkeratotic hand eczema (Follicular ECZ.)
- 3-dry palmar eczema (Housewife ECZ)
- 4-finger tip eczema
- 5-ring eczema
- 6-localized thumb ECZ (لا يمتد إلى بقية الأصابع)
- 7-discoid eczema
- 8-chronic acral dermatitis (Hand ECZ. + ↑ IgE)
- 9-apron eczema (زيت الطبخات)
- 10-gut eczema (بزاز) → blood, fat (ICD)
- 11-other patterns (eg. patchy vesiculosquamous)

والأهم من ذلك
أنه قد يكون
أداة تشخيصية
(وخاصة في
الحالات الغريبة)

1. جواتي يا سترار مع الغسل وقطع الأكل - ومعالجته بمرحلة

- Treatment
- 2 Potent or super potent Cs for 2-3 wks → Week-end
 - 3 + 5 ds / wk → Weak potent Cs ~ Tacrolimus
 - 4 Emollients: مرطبات يومية باستمرار لا يتوقف